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# **Report of The Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario**

Volume One

**J. Stefan Dupré**  
Chairman

**J. Fraser Mustard**  
Commissioner

**Robert J. Uffen**  
Commissioner

Donald N. Dewees  
Director of Research

John I. Laskin  
Legal Counsel

Linda B. Kahn  
Executive Co-ordinator





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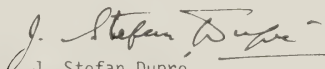
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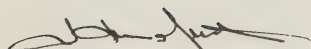
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April 1984

May it please Your Honour:

By Order-in-Council 1243/80, dated the 29th day of April, 1980, we were constituted a Royal Commission under the Public Inquiries Act to inquire into matters of health and safety arising from the use of asbestos in Ontario. We have completed our inquiry and beg to submit the accompanying Report.

  
J. Stefan Dupré  
Chairman

  
J. Fraser Mustard  
Commissioner

  
Robert Uffen  
Commissioner



## Order-in-Council

O.C. 1243/80

Copy of an Order-in-Council approved by Her Honour the Lieutenant Governor, dated the 29th day of April, A.D. 1980.

The Committee of Council have had under consideration the report of the Honourable the Minister of Labour wherein he states that

WHEREAS, the Government of Ontario believes that it is in the public interest that ample opportunity is afforded for full public input into the assessment of the matter of health and safety arising from the use of asbestos in Ontario and that the benefit of the best medical, scientific and other expert advice, both from within the government and from the community at large is made available in arriving at conclusions as to the need for further remedial action, and

WHEREAS, the Government has reached the conclusion that these aims can best be achieved by means of a public inquiry instituted pursuant to the provisions of The Public Inquiries Act, 1971, Chapter 49;

The Honourable the Minister of Labour therefore recommends that a Commission be established pursuant to the provisions of The Public Inquiries Act, 1971, Chapter 49, to study and report to the Minister of Labour on the matter of health and safety arising from the use of asbestos in Ontario and:

1. To investigate all matters relating to health and safety arising from the use of asbestos in Ontario;
2. To identify the relevant data related to asbestosis, mesothelioma and other diseases and health hazards of persons working with or exposed to asbestos in Ontario;

3. To review the present basis for Workmen's Compensation Board awards as they relate to occupational health matters affecting workers exposed to asbestos, including any special programs dealing with the rehabilitation of such workers;
4. To make such recommendations in relation to the above as the Commission deems appropriate.

The Honourable the Minister of Labour further recommends that the following persons be appointed members of the Commission:

Dr. Stefan Dupré  
Dr. Fraser Mustard  
Dr. Robert Uffen

and that Dr. Stefan Dupré be designated Chairman of the Commission.

And the Honourable the Minister of Labour further recommends that all government ministries, boards, agencies and commissions assist the commission to the fullest extent in order that it may carry out its duties and functions,

AND THAT the commission have authority to engage counsel, expert technical advisors, investigators and other staff as it deems proper at rates of remuneration and reimbursement to be approved by the Management Board of Cabinet in order that a complete and comprehensive report may be prepared and submitted to the Government,

AND THAT the Ministry of the Attorney General will be responsible for providing administrative support to the commission.

The Committee of Council concur in the recommendations of the Honourable the Minister of Labour and advise that the same be acted on.

Certified,

(signed)    Deputy Clerk, Executive Council



## Acknowledgements

A report of this size and complexity is by necessity the product of many hands. As Commissioners, we alone are responsible for its content, but our indebtedness for its existence encompasses many individuals.

The three members of our senior staff, Dr. Donald N. Dewees, Director of Research; Mr. John I. Laskin, Legal Counsel; and Miss Linda B. Kahn, Executive Co-ordinator, were to us full partners in every sense save responsibility for the end product. Their selfless devotion and dedicated professionalism infused every stage of our work. As the only partner who was engaged in this enterprise on a full-time basis, Miss Kahn deserves special gratitude for orchestrating us all and for bearing enormous administrative and editorial burdens with gracious aplomb and awesome efficiency.

We gratefully acknowledge the devoted services of the members of our research, administrative, and secretarial staff, whose names appear in Appendix G at the end of this Report. In particular, we thank Mrs. Phyllis D. Bartley, who served simultaneously as Miss Kahn's right hand and as our senior secretary from the day we were appointed. To our all-purpose researcher, Mr. Ronald J. Daniels, and our librarian, Mrs. Patricia S. Rubin, we also express special gratitude.

For the knowledge that infused our deliberations, we owe an enormous debt to the Canadian, American, British, and Swedish scientists and officials who appeared before us as expert witnesses (see Appendix B); to the authors and reviewers of our research studies (see Appendix F); and to our consultants (see Appendix H). Among the latter, we wish to cite the especially generous contribution in time and effort of Professors David C.F. Muir, Robin S. Roberts, and Neil Rowlands.

Throughout the many days on which we heard sworn testimony, we drew invaluable assistance from the individuals who acted as the representatives of the several parties granted formal standing by this Commission

(see Appendix A). Their examination and cross-examination of witnesses added materially to the quality of the evidence before us. We express our gratitude to these representatives, Ms. Linda Jolley and Messrs. Jean Bazin, Orlando Buonastella, Phillipe Casgrain, Ed Cauchi, Rick Evans, Brian Gibson, Timothy S. Hardy, Thomas R. Lederer, Nick McCombie, James McNamee, Arthur F. Sampson III, David K.L. Starkman, Daniel Ublansky, and Edward W. Warren.

Finally, we warmly thank the many persons who appeared before us during the informal phases of our hearings. The extent of their assistance is visibly apparent in the text of our Report and is cited in its documentation.

J.S.D.

J.F.M.

R.J.U.

# **Part I**

## **Introduction**





# Chapter 1 The Report of The Royal Commission on Asbestos: An Overview

## A. Another Royal Commission

Was this Royal Commission really necessary?

Perhaps deservedly, perhaps not, royal commissions in Canada have acquired a reputation for being slow, elaborate, and expensive means of investigating subjects of varying degrees of public importance and controversy. The range of these subjects has been almost infinite: they have been as specific as the affairs of a lone person and as vast as the economic prospects of the entire realm. We were appointed by the Government of Ontario to deal with matters of health and safety arising from the use of a single substance in this province: asbestos. That asbestos can be a devastatingly serious health hazard was widely known many years before this Commission was appointed in April of 1980. We were hardly needed to confirm this fact.

This stark fact has understandably aroused widespread public apprehension. Asbestos is everywhere: it is in the air we breathe, the water we drink. It has been used for generations because of its superb fire- and acid-resistant qualities. Canada is the largest producer of asbestos in the western world. World-wide, asbestos has brought disease and death to literally thousands of individuals who have been exposed to its microscopic fibres as miners and millers, manufacturing workers, insulators, and members of other occupations. It turns out that these individuals, as conveyed by the title of a well-known book on the subject, were literally *Dying for a Living*.<sup>1</sup> What are the implications for the continued use, if any, of asbestos

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<sup>1</sup>Lloyd Tataryn, *Dying for a Living* (Toronto: Deneau and Greenberg Publishers Ltd., 1979).

in industry? More generally, does this known workplace killer threaten unsuspecting individuals in office buildings, airports, schools?

Necessary or otherwise, this Commission had no shortage of attention. An initial public meeting, convened in October of 1980, attracted about 400 individuals, and this attendance was almost matched at a second public meeting six weeks later. These meetings had been announced in advertisements placed in 81 daily and weekly newspapers throughout Ontario. The advertisements also publicized the existence of a brochure which was available on request and which described our plan of activities. This brochure, which was also mailed on an unsolicited basis to persons likely to have an interest in our terms of reference, included a return postcard on which the recipient could indicate the desire to be placed on the Commission's mailing list or declare the intention to make a written or oral submission. Over 2,000 individuals and organizations placed themselves on our mailing list and were thereby kept abreast of all hearing dates and of our research programme. During the week of February 16 through 20, 1981, we sat morning, afternoon, and evening hearing oral submissions in Toronto in a setting as informal as the intermittent glare of television lights permitted. The presenters ranged from seriously ill workers and lone widows to leaders of major business, labour, and government organizations. Other such informal hearings ensued, in Windsor on March 27, 1981 and Toronto on June 8, 1981. But royal commissions, at least in Ontario, involve more than informal hearings; this is perhaps not least of the reasons why they are reputedly slow and ponderous.

Pursuant to section 5(1) of the *Public Inquiries Act*,

A commission shall accord to any person who satisfies it that he has a substantial and direct interest in the subject-matter of its inquiry an opportunity during the inquiry to give evidence and to call and examine or to cross-examine witnesses personally or by his counsel on evidence relevant to his interest.<sup>2</sup>

In October of 1980, we gave notice that persons who wished to apply for standing pursuant to section 5(1) should so indicate by January 31, 1981, and we thereupon granted standing to the twenty parties listed in Appendix A at the end of this Report. We decided further that one of these parties, the Asbestos Victims of Ontario, should be granted public monies to defray the costs of its representation. The Asbestos Victims of Ontario is a group composed of former asbestos workers, their spouses, or widows.

Over the ensuing months, the several representatives of the parties-with-standing met regularly with our legal counsel to develop, by consensus,

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<sup>2</sup>R.S.O. 1980, c. 411.



the names of witnesses who should be invited to testify. The response rate to our invitations turned out to be almost 100%. The names of our witnesses are listed in Appendix B. These 53 men and women came to give sworn testimony not only from Ontario and elsewhere in Canada but from Sweden, the United Kingdom, and the United States. Most underwent one and sometimes two full days of examination and cross-examination. About half our witnesses were scientists, constituting an international who's who of experts on the health effects of asbestos. The taking of formal testimony consumed 53 hearing days, concentrated mainly during the summer months of 1981 and 1982.

While our hearings were in progress, we undertook, either personally or through our staff, to acquire yet additional knowledge through direct observations and informal contacts. This involved, by way of illustration, visits to some Quebec asbestos mines, to the Mount Sinai School of Medicine in New York, to certain Ontario manufacturing establishments, and to some asbestos removal projects. We also undertook a research programme to improve our understanding of the issues before us. This programme yielded two background papers and ten book-length research studies, listed in Appendix F, which we published and released for assessment and criticism as they were completed. The subjects covered by these studies ranged from the technical problems of measuring asbestos levels to the matter of workers' compensation for asbestos-related disease in Ontario. Because the studies began to appear in February of 1982, the information they divulged and the perspectives they provided became a useful adjunct to our remaining hearings. Our last day of formal hearings was August 24, 1982. After allowing the parties-with-standing sufficient time to review the evidence, we returned to the hearing room for one final occasion: the presentation of final submissions by these parties on January 28, 1983. Otherwise, we sat closeted in deliberations with our director of research, our legal counsel, and our executive co-ordinator, surrounded by our 12 research studies and background papers, our 88 written submissions, and our 8,378 pages of oral presentations and sworn testimony.

This Report is the outcome of those deliberations. Both its length and its prose — a literary genre we call “commissionese” — ensure that it will be read from cover-to-cover by precious few. In this chapter, we shall attempt to distil our findings to their bare essentials. Readers who — from anger or delight, bemusement or curiosity, or any other motive — desire elaboration need only glance at the footnotes to find the particular chapters or sections of chapters that divulge our information base and our detailed reasoning. We have been prescriptive as well as analytical; we shall therefore convey the gist of our prescriptions as this chapter unfolds and provide, at its end, the text of each of the 117 formal recommendations that appear in the body of the Report.

## B. Health Effects of Asbestos

Residents of Ontario have more than the normal run of reasons to share the international feelings of apprehension that asbestos has aroused. This province is the scene of what we document in this Report to be a world-class occupational health disaster: the Johns-Manville plant in Scarborough, in the Municipality of Metropolitan Toronto. This plant, between 1948 and 1980, manufactured asbestos-cement pipe, using a mixture of two kinds of asbestos: chrysotile and crocidolite. At various times during its existence, this plant also manufactured asbestos-cement board, using only chrysotile, and asbestos insulation materials, using chrysotile and a third kind of asbestos called amosite.<sup>3</sup>

As of 1983, the death toll from asbestos exposure in this plant, as measured by the number of claims awarded by the Ontario Workers' Compensation Board, was 68.<sup>4</sup> This lone plant, whose annual employment never exceeded 714 workers, has already occasioned more deaths from industrial disease than the entire Ontario mining industry, which annually employs over 30,000 workers, occasions from industrial accidents in an average four-year period.

The death toll at this plant, which closed in 1980, offers harsh testimony to the nature of long-latency disease.<sup>5</sup> It has mounted gradually and inexorably because individual deaths are separated from the beginning of the exposures that caused them by some 10 to 30 or more years. The dimensions of the disaster have therefore been growing over time; for example, between August 1981 and August 1982, the middle year in the existence of this Commission, 5 more ex-employees died of mesothelioma, a rare cancer that is specifically associated with asbestos exposure. It is a tragically safe assumption that, among those who worked in this plant, asbestos-related deaths will continue to occur, and that hence the disaster has yet to run its course.

Asbestos-related deaths are from mesothelioma. They are from lung, gastrointestinal, and laryngeal cancers which, while they arise from a variety of sources, can also be caused by asbestos. They are from asbestosis. The last of these diseases, as its name suggests, is asbestos-specific. It is a form of lung fibrosis which can itself be fatal, but more commonly

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<sup>3</sup>For a description of the different types of asbestos, see Chapter 2, Section B.

<sup>4</sup>For a full account of the disease and mortality experience in this plant, see Chapter 3, Section A. For an account of the cost of compensating the plant's victims, see Chapter 14, Section C. A special rehabilitation effort undertaken for workers at this plant by the Workers' Compensation Board is discussed in Chapter 14, Section A.

<sup>5</sup>For a description of asbestos-related diseases and the phenomenon of long latency, see Chapter 2, Section D. For a further discussion of the time interval between the initial exposure to asbestos and the clinical manifestation of disease, see Chapter 5, Section E.4.

makes its victims susceptible to death from related causes and, most significantly, is an irreversible, disabling, normally progressive condition. By the time the Johns-Manville plant closed in 1980, 113 of its exposed workers had been awarded disability pensions by the Workers' Compensation Board for their varying degrees of medical impairment occasioned by asbestosis.

The asbestos-induced disaster at this plant ranks with the worst that have been recorded in the international epidemiological literature on asbestos. It places the name Scarborough on an unenviable list with Charleston, South Carolina; Rochdale, England; and a handful of other places.<sup>6</sup> The Scarborough plant accounts for half of all the asbestos deaths and disabilities that have been compensated by the Ontario Workers' Compensation Board. The remainder have been occasioned by exposure in a wide variety of work situations and industrial processes, for example, war-time gas mask manufacturing, brake manufacturing, and shipbuilding. The Ontario employers whose workers suffered asbestos disease and death are spread throughout the province; excluding Johns-Manville, only two have given rise to more than 5 awarded claims.<sup>7</sup>

There is indeed reason to be apprehensive about asbestos in Ontario. But what is this Commission's "bottom-line" on the nature of the health hazard posed by asbestos? Our "bottom-line" can be reduced to the following propositions:

1. Asbestos diseases offer a classic instance of the phenomenon that is called a dose-response relationship. The greater the exposure of an individual to asbestos (the dose), the greater the likelihood of disease (the response). Conversely, the lower the exposure of an individual to asbestos, the lower the likelihood of disease.<sup>8</sup> However, at low levels of exposure, the assessment of disease risk is bedevilled by uncertainty. The uncertainty arises from a number of factors including difficulties of measurement, limitations of epidemiology, and lack of data.<sup>9</sup> Accordingly, prudence dictates the assumption that any exposure involves some risk of disease.

2. The overwhelming weight of the scientific evidence is that disease results from breathing (inhaling) asbestos fibres. There is no substantial evidence that disease can result from swallowing (ingesting) asbestos fibres

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<sup>6</sup>For a comparative analysis of disease and death among asbestos workers in various plants throughout the world, see Chapter 5, Section B.

<sup>7</sup>For an account of the volume of compensated disease and death claims among various Ontario employers, see Chapter 3, Section A.

<sup>8</sup>For a discussion of dose-response relationships for the asbestos-related diseases, see Chapter 5, Section E and Chapter 7, Section C.

<sup>9</sup>For a discussion of these factors, see Chapter 4, Section D.



and only the most speculative of hypotheses on how ingestion could give rise to disease.<sup>10</sup>

3. The asbestos fibres which are most likely to cause adverse health effects when inhaled are long and thin. "Length" and "diameter" are, of course, relative phenomena: fibres are measured in microns, one micron being one-millionth of a metre. The hazardous asbestos fibres are those which would be longer than 5, perhaps longer than 8 microns, and thinner than 1.5 or perhaps 0.25 microns.<sup>11</sup>

4. There is strong evidence that crocidolite and amosite fibres tend to be more hazardous than chrysotile fibres, primarily because they are more likely to conform to the most hazardous length and diameter and secondarily because they are more likely to become airborne and hence to be respirable. It is also possible that crocidolite and amosite are more hazardous because of their chemical composition, but this possibility rests in the realm of speculation.<sup>12</sup>

5. All fibre types can cause all asbestos-related diseases, but mesothelioma is most likely to result from crocidolite exposure, has a strong association with amosite exposure, and has a weak association with chrysotile exposure.<sup>13</sup>

6. Whatever their type, the likelihood that asbestos fibres of the most hazardous dimensions are respirable is strongly a function of the individual process in which asbestos is being used. Thus, for example, the manufacture of brake linings, which involves drilling and grinding, is much less likely to generate fibres of hazardous dimensions than textile manufacturing, which involves spinning and weaving.<sup>14</sup>

7. The individuals who have been afflicted with asbestos-specific diseases have almost invariably been occupationally exposed to asbestos. However, there is incontrovertible evidence that mesothelioma has afflicted individuals who shared the domicile of an asbestos worker at the time this

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<sup>10</sup>For an account of the biological evidence as to the ingestion of asbestos fibres, see Chapter 4, Section B.2. For a discussion of the epidemiological evidence, see Chapter 5, Section I.

<sup>11</sup>For an analysis of the importance of fibre dimension, see Chapter 4, Section B and Chapter 5, Section D.

<sup>12</sup>For a discussion of the evidence which indicates that crocidolite and amosite tend to be more hazardous than chrysotile, see Chapter 5, Section C. For an account of the significance of fibre dimension and the possible significance of fibre chemistry, see Chapter 5, Section D.

<sup>13</sup>For an account and an analysis of the evidence concerning the incidence of mesothelioma in relation to different fibre types, see Chapter 5, Sections C.4, C.6, and D; and Chapter 7, Sections C and F.

<sup>14</sup>For a review of the health experience of workers in various processes using asbestos, see Chapter 5, Section B and Chapter 7, Section C. For a discussion of the effects of process on fibre dimension and the consequent health effects, see Chapter 5, Section D and Chapter 7, Sections C and F. For a description of different industrial processes, see Chapter 6, Section C.

worker was exposed. There is weak evidence that individuals may have contracted disease from exposure to asbestos in the neighbourhood of asbestos plants.<sup>15</sup> We have found no evidence that disease afflicts individuals who breathe asbestos in the outdoor air or inhale it as occupants of asbestos-containing buildings.<sup>16</sup>

8. Asbestosis is a disease which is attributable solely to occupational exposure to asbestos. There is substantial evidence that at a very low level of occupational exposure to asbestos, the fibrotic process in the lungs, if indeed it can even be initiated, will not develop to the point of producing asbestosis at the level of clinical diagnosis or of producing any manifestation that would cause even the mildest discomfort to an individual.<sup>17</sup>

9. There exists no relationship between smoking and mesothelioma. Asbestosis cannot be caused by smoking, but there is evidence that smoking can accelerate the pace at which the fibrotic process develops. Finally, while asbestos alone can cause lung cancer, there is clear evidence that asbestos and smoking interact so synergistically in causing this disease that smoking coupled with asbestos work is like pouring gasoline on a fire.<sup>18</sup>

We have made a number of other findings on the health effects of asbestos, but the above can be taken to be the highlights. What are the implications of these findings? We answer this question first by examining asbestos in fixed place industry, then asbestos in buildings, and finally asbestos elsewhere.

## C. Asbestos in Fixed Workplaces

Fixed workplaces, which in the case of asbestos mean mining and manufacturing, involve, for regulatory purposes, a distinct compartment of asbestos-related activities for several reasons. First, these activities are optional; they could be banned if asbestos exposure were deemed to involve unacceptable risks. Second, these activities, being confined to fixed workplaces, are amenable to work practices and engineering controls which reduce the concentration of asbestos fibres to which workers are exposed. Third, and again because these activities are in fixed workplaces, it is possible to monitor, in an ongoing manner, the concentration of asbestos fibres in the air. The prevailing measurement technique involves: (i) air sampling by what is known as the membrane filter method; (ii) counting the number of fibres captured from each air sample with the use of an optical micro-

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<sup>15</sup>For an elaboration of these points, see Chapter 5, Section E.

<sup>16</sup>See Chapter 11, Section C and Chapter 9.

<sup>17</sup>For a discussion of this evidence, see Chapter 5, Section E.3.

<sup>18</sup>For a discussion of the relationship between smoking, asbestos exposure, and disease, see Chapter 5, Section F.

scope; and (iii) expressing the number of fibres detected on the basis of so many fibres per cubic centimetre of air [fibre per cubic centimetre (f/cc)].<sup>19</sup>

In accordance with the provisions of the Ontario *Occupational Health and Safety Act*, asbestos is a designated substance and is covered by the Regulation Respecting Asbestos. This Regulation enjoins fixed place employers to “take all necessary measures and procedures” to ensure that the exposure of a worker to airborne asbestos “is reduced to the lowest practical level” and stipulates control limits which are not to be exceeded. These limits are 1 f/cc for chrysotile, 0.5 f/cc for amosite, and 0.2 f/cc for crocidolite. The Regulation extends to all fixed place, asbestos-using industry and does not discriminate among industrial activities.

Historically in Ontario, asbestos has been mined and used in a wide range of manufacturing processes. Mining, which involves chrysotile asbestos, is for the time being a trivial to non-existent activity; manufacturing, to the best of our knowledge, is currently confined to the use of chrysotile asbestos and is in the main devoted to friction products, including automotive brakes.<sup>20</sup>

What is the risk to which workers are exposed from these and, for that matter, any other asbestos-using activities that involve fixed place industry? Asbestos is one of the most thoroughly studied industrial carcinogens in the world. There accordingly exists a data base from which to estimate the risk of disease at different levels of occupational exposure. We have used models of disease incidence and data from a number of epidemiological studies of workers exposed to asbestos to forecast the possible disease risk that may result from exposure to varying concentrations of all types of asbestos dust in varied manufacturing processes.<sup>21</sup>

On the basis of this analysis, we conclude that we cannot condone any manufacturing activity that involves the use of crocidolite or amosite asbestos.<sup>22</sup> The use of these two types of asbestos in Ontario should,

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<sup>19</sup>For a detailed description and analysis of the membrane filter method, see Chapter 7, Section B.

<sup>20</sup>For a description of asbestos mining in Ontario, see Chapter 6, Section F; manufacturing activity is described in Chapter 6, Section B.

<sup>21</sup>These estimates are developed in Chapter 7, Section C. A technical description of the risk assessment model is contained in the Appendix to Chapter 7.

<sup>22</sup>For the criteria we have developed to assess when a substance should be subject to control limits, when it should be prohibited, and the relationship of a substance to the availability of substitutes, see Chapter 7, Section E. The feasibility and cost of asbestos control methods are discussed in Chapter 6, Section C. For a discussion of substitutes and their health effects, see Chapter 6, Sections D and E.



therefore, be prohibited indefinitely.<sup>23</sup> As for chrysotile, it is necessary to distinguish the industrial processes in which asbestos is being used. The control limit that the Regulation Respecting Asbestos currently applies to all chrysotile processes is 1 f/cc. If this control limit is rigorously observed so that, at the level of an 8-hour time-weighted average, it is rarely exceeded, we calculate that the average level of exposure to which workers will be subjected is no greater than 0.5 f/cc.<sup>24</sup> Our data base permits us to calculate the disease risk encountered by workers in the following industrial processes: mining and milling; general manufacturing, excluding textiles and cement products; textile manufacturing; and cement product manufacturing. We find in the cases of chrysotile mining and milling and of general chrysotile manufacturing that the disease risk associated with chrysotile exposure under a 1 f/cc control limit, effectively enforced, involves a projected mortality rate well below the mortality rate that results from industrial accidents in all Ontario manufacturing. It therefore falls well within the bounds of a societally acceptable industrial risk.<sup>25</sup>

Textile manufacturing with chrysotile is starkly different. Under a 1 f/cc control limit, effectively enforced, the estimated risk from mortality in this asbestos-using process exceeds the risk of mortality from accidents in manufacturing and approaches or passes the risk of mortality from fatal accidents in general Ontario mining activity. This activity, with respect to industrial accidents, has long been one of the most hazardous in Ontario. To reduce the risk of death from disease in chrysotile-using textile product manufacturing to that in other chrysotile-using processes would require a control limit of 0.04 f/cc, far lower than any limit which, given prevailing measurement technology, could be enforced. We find that this chrysotile-using process therefore cannot be condoned in Ontario, and that it should be banned until it were to become possible to satisfy the Ministry of Labour that its introduction would involve risk of no greater significance than that posed by mining and general chrysotile manufacturing.

The risk level we associate with chrysotile mining and general manufacturing may be lower than the accident risk in manufacturing, but it is still significant and is a disease risk borne by asbestos workers who, like all manufacturing employees, run the risk of fatal industrial accidents. We, therefore, view the following as essential if chrysotile use is to be tolerated

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<sup>23</sup>See Chapter 7, Section F.3. The ban we recommend takes the form of outright prohibition by the Ministry of Labour which could only be lifted if health risks were greatly reduced. In the case of amosite, this would necessitate a currently unattainable exposure maximum of 0.1 f/cc. (See Recommendation 7.14.) In the case of crocidolite, the exposure maximum, if any, would have to be 0.02 f/cc. (See Recommendations 7.12 and 7.13.)

<sup>24</sup>For the basis of this calculation, together with an extensive discussion of sampling and measurement errors, sampling frequency, and related issues, see Chapter 7, Section B.

<sup>25</sup>For an extensive discussion of this point and of the points summarized in the balance of this paragraph and the following paragraph, see Chapter 7, Section F.



at a control limit of 1 f/cc: (i) All workers must be fully informed of the risk they are facing, as the Regulation Respecting Asbestos contemplates. (ii) The Regulation must be strictly enforced so that the control limit of 1 f/cc in fact ensures that actual average exposure will be 0.5 f/cc. (iii) The Regulation, the *Occupational Health and Safety Act*, and the practices and organization of the Ministry of Labour must all be modified to give assurance to the Government and Legislature, the public, and above all the workers concerned that the regulation of chrysotile asbestos is indeed being implemented effectively.<sup>26</sup>

We have made formal recommendations in this regard which range from developing more representative air sampling procedures to increasing worker participation in the enforcement and monitoring process. We single out one of these recommendations, which is that the Ministry of Labour should create a Designated Substances Enforcement Unit to supplement the enforcement role of its inspectorate in workplaces where designated substances are present. Other than for its Director, whose qualifications would include training in investigative techniques, the Unit would not require the engagement of new Ministry staff but would instead involve selecting interdisciplinary teams of professionals from the Occupational Health Branch, assembled by the Director, for the purpose of making unannounced visits to plants using designated substances.<sup>27</sup>

## D. Asbestos in Buildings

In Ontario, as in a number of other jurisdictions, asbestos was widely used until 1973 as sprayed insulation material for fire protection and acoustical purposes and in pipe and boiler insulation.<sup>28</sup> Accordingly, it is present in a large number of multi-storey buildings, including offices, schools, factories, and air terminals. The types of asbestos that are generally present are not only chrysotile but amosite.

Understandably, the association of chrysotile and amosite asbestos with buildings has created enormous public apprehension. Is this apprehension warranted? Our response to this question comes in two parts. First, asbestos is indeed a potentially serious hazard to workers who are directly engaged in such construction-related activities as asbestos removal and building demolition, or who are involved in a wide variety of activities in

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<sup>26</sup>For an extensive discussion of the Regulation Respecting Asbestos, the internal responsibility system, and enforcement, see Chapter 8.

<sup>27</sup>See Chapter 8, Section E.1.

<sup>28</sup>For a discussion of the application and characteristics of sprayed insulation and pipe and boiler insulation, the buildings in which these are found and the types of asbestos used, see Chapter 9, Section B.

the realm of building maintenance.<sup>29</sup> Second, the inhalation of chrysotile and amosite fibres by building occupants almost never poses a health hazard, save perhaps if particularly elevated exposure is occasioned by the disturbance of asbestos, especially in removal projects.<sup>30</sup>

Let us immediately elaborate on our second finding. It is essentially in line with one made by the United Kingdom Advisory Committee on Asbestos in 1979. Notwithstanding this British finding, it seems evident that public apprehension about the risk which asbestos poses to building occupants is not easily allayed. If this is so, it speaks strongly in favour of more pointed and co-ordinated communication by public agencies. In particular, we find that a major cause of public apprehension must be attributed to the failure of government agencies involved in asbestos regulation to communicate clearly with respect to the all-important factor of measured levels of asbestos in building air. Ontario is itself a classic example of a jurisdiction in which two different agencies have utterly failed in co-ordinating their communication. Thus, in Ontario, the Ministry of Labour has promulgated well-publicized control limits for fixed workplaces. As we have seen, the chrysotile control limit is 1 f/cc. Meantime, the Ministry of the Environment has a guideline for asbestos in the ambient air (generally extrapolated to building air) of 0.04 f/cc. At first blush, it would appear that the Environment guideline is 1/25 that of the Labour control limit. But this is not true. The Labour control limit of 1 f/cc applies in a setting where the number of fibres in air samples is counted with the use of an optical microscope. By contrast, the low fibre concentration envisioned by the Environment guideline involves fibre counting by electron microscopy.<sup>31</sup> The electron microscope detects a great many fibres too thin to be seen through the optical microscope. Accordingly, a count of asbestos fibres in building air using electron microscopy will find more fibres, to the extent of an order of magnitude or perhaps 10 times as many fibres as a count through the optical microscope.<sup>32</sup> It follows that the Environment guideline of 0.04 f/cc has the effect of being less like 1/25 and more like 1/250 the Labour control limit.

We find that outdoor airborne asbestos fibre concentrations are often less than 0.001 f/cc measured using an electron microscope, and hardly ever reach 0.01 f/cc.<sup>33</sup> The optical equivalent of these concentrations would be an order of magnitude smaller. Perhaps more important, we conclude

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<sup>29</sup>For a discussion of the asbestos exposures to which building workers have been and can potentially be subjected, see Chapter 9, Sections B and D.

<sup>30</sup>For an analysis of the exposures of building occupants to asbestos, see Chapter 9, Section D.

<sup>31</sup>For a discussion of the ambient air guideline, see Chapter 11, Sections C.4 and C.5.

<sup>32</sup>For a discussion of the relationship between optical fibre counts and electron microscope fibre counts, see Chapter 7, Section B and Chapter 11, Section C.1.

<sup>33</sup>For a discussion of data on outdoor asbestos concentrations, see Chapter 11, Section C.2.

that the air in buildings with sprayed asbestos-containing insulation usually averages less than the equivalent of an optical fibre count of 0.001 f/cc, and the highest readings would rarely exceed 0.01 f/cc. Acknowledging that building air is contaminated not only by chrysotile but by the more hazardous amosite fibres, it is possible to estimate the risk to building occupants who are exposed to a mixture of chrysotile and amosite fibres at the level of 0.001 f/cc. The risk that an individual who had been exposed to asbestos in a building for, say, 10 years would die of an asbestos-related disease is less than 1/50 the risk that death would come from commuting 10 miles by auto to and from that building daily for the same period. At this level, we deem the risk which asbestos poses to building occupants to be insignificant and therefore find that asbestos in building air will almost never pose a health hazard to building occupants.<sup>34</sup>

The same, however, is not true with respect to workers engaged in demolition, asbestos removal, or building maintenance activities. Such workers may become exposed to asbestos levels that greatly exceed the control limits which apply in fixed place industry.<sup>35</sup> The exposure involved is to amosite as well as chrysotile asbestos. This exposure, in turn, may come in short, intense bursts that can overcome the normal defence mechanisms of the human lung. Where demolition, removal, and certain repair activities are involved, the workplace is not fixed but instead resembles that of the typical construction project. It follows that worker protection can better be enforced by specifying work procedures than by using air monitoring to enforce control limits, and we prescribe elaborately in this regard.<sup>36</sup> The magnitude of the potential risk from exposure in building demolition is such that we specifically prescribe that all friable asbestos should be removed from buildings prior to demolition.<sup>37</sup>

Since 1979, numerous Ontario school boards, with some \$26 million in financial assistance from the provincial government, have made massive efforts to remove asbestos from schools.<sup>38</sup> This has been in direct response to parental apprehension concerning the health of their children. There is substantial evidence that the possibility of developing mesothelioma is linked to the amount of time that has elapsed since first exposure, meaning that the younger a person's age when initially exposed to asbestos, the

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<sup>34</sup>For the analysis of the health risk posed by asbestos exposure to building occupants, see Chapter 9, Section E.

<sup>35</sup>For data on the exposure of building workers, see Chapter 9, Sections B and D; and Chapter 10, Section C.

<sup>36</sup>For an explanation of regulation by procedure in contrast to regulation by control limit, see Chapter 10, Section A.

<sup>37</sup>See Chapter 10, Section C.3. For an analysis which compares the cost of asbestos removal while a building is still in use to the cost of asbestos removal just prior to demolition, see Chapter 9, Section E.

<sup>38</sup>For a discussion of the asbestos control programme in Ontario schools, see Chapter 9, Section E.



greater the lifetime risk of developing mesothelioma.<sup>39</sup> But acknowledging this fact, and even allowing for the hypothesis that the very young might be more susceptible to asbestos disease,<sup>40</sup> the health risk to children remains insignificant because the level of exposure in asbestos-containing schools has in general been so low. The exceptions are found in those instances where loose asbestos is being actively disturbed or is falling from ceilings. It follows that, with these exceptions, the programme for removing asbestos from all asbestos-containing schools was not justified by the health risk posed to students. In some cases, the asbestos control work may have been justified in order to protect building renovation, maintenance, or custodial workers from exposure to asbestos if their work would have disturbed asbestos insulation. But neither the scale nor the pace of the school programme was warranted by the risk posed by most asbestos-containing schools to occupants or workers. If anything, the scale and pace of the programme significantly *increased* the risk to some workers directly engaged in control projects. Crash programmes invariably mean that inexperienced contractors and personnel will enter the field. A number of asbestos-control projects in schools were conducted in a manner which, according to the evidence before us, may have generated significant risk for the workers involved. There is no question, however, that the crash nature of the asbestos control programme in schools was occasioned by a climate of public apprehension; the actions of school boards and of the Ministry of Education, paralleled in other jurisdictions, show them to be responsive to public demand.

## E. Asbestos Elsewhere

We are all exposed to asbestos because it is in the water we drink and the air we breathe. Counts of up to 4 million fibres per litre of drinking water have been found in southern Ontario municipalities (e.g., Sarnia and Metropolitan Toronto) and of up to 22 million fibres per litre in northern Ontario municipalities (Thunder Bay). The fibres are almost invariably extremely short, that is, below 1 micron in length.<sup>41</sup> The small dimension of these fibres, coupled with our finding that asbestos disease is occasioned by inhalation rather than ingestion, leads us to conclude that asbestos in drinking water is not a health hazard.<sup>42</sup> The same conclusion applies to asbestos in food and drink,<sup>43</sup> and we indeed recommend that a ban which the

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<sup>39</sup>For a discussion of the hypothesis that mesothelioma is time-dependent, see Chapter 5, Section E.4.

<sup>40</sup>See Chapter 5, Sections E.4 and H.

<sup>41</sup>For a description of the levels and dimensions of asbestos fibres in drinking water, see Chapter 11, Sections B.1 and B.2.

<sup>42</sup>For our analysis of the health effects of ingesting asbestos fibres, see Chapter 5, Section I.

<sup>43</sup>For a discussion of asbestos in food and beverages, see Chapter 11, Section B.3; the topic of asbestos in drugs is discussed in Chapter 11, Section B.4.



Liquor Control Board of Ontario has imposed on the use of asbestos filters in the production of beer, wine, and liquor should be lifted. (Ontario drinkers who remain apprehensive may wish to note that this ban was never effectively enforced on imported alcoholic beverages.)

We review asbestos in the outdoor air and note the prevalence of very short fibres and of low counts (a maximum of 0.0084 f/cc of all lengths) even in the vicinity of a Toronto expressway ramp, where vehicle braking action might be expected to release fibres.<sup>44</sup> We conclude that an ambient air quality objective such as the 0.04 f/cc guideline of the Ministry of the Environment remains important, not so much because of any health hazards that might be posed at such levels of exposure, but so that any unusual level of contamination can be identified and its source sought out. As for the adequacy of the 0.04 f/cc guideline, we recommend that the Ministry of the Environment should maintain it until internationally accepted methods for outdoor air measurement are adopted and permit a discerning review of its numerical level.

With respect to asbestos-containing consumer products, we express substantial concern over the pattern of regulation.<sup>45</sup> This pattern has been, in a word, erratic. Thus, for example, Health and Welfare Canada recommended that the use of asbestos in hand-held hairdryers should be discontinued although tests showed fibre release equivalent to normal background levels in outdoor air. Meantime, free-form asbestos, which may occasion significant fibre release, is unregulated and currently available at hardware stores throughout the country. As in many other aspects of Canadian life, the federal and provincial levels of government share jurisdiction over the regulation of consumer products. We prescribe that the Government of Ontario should take steps, preferably through federal-provincial collaboration, to sort consumer products into three categories. The first would be those that can release significant levels of asbestos fibres in normal use (e.g., loose-fill asbestos insulation); the second, those that possess the potential of releasing fibres from cutting or sanding or as a result of degradation (e.g., asbestos-cement sheet or asbestos gloves); the third, those products in which asbestos is sealed off or encapsulated (e.g., most appliances which contain asbestos insulation). The sale of products in the first category should be prohibited (unless there is no adequate substitute or the product is exclusively for the use of a manufacturing enterprise governed by the Regulation Respecting Asbestos). Products in the second category should be labelled and instructions made available on their safe use. Products in the third category should not be subject to regulation.

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<sup>44</sup>For a description of asbestos levels in the outdoor air, and for an elaboration of the points summarized in the balance of this paragraph, see Chapter 11, Section C.

<sup>45</sup>For an elaboration of this and the points summarized in the balance of this paragraph, see Chapter 11, Section A.

Finally, we single out the disposal of asbestos wastes as a source of concern, not so much because of any resulting contamination of outdoor air but to protect waste disposal workers from exposures that could pose a significant risk to their health.<sup>46</sup> Until it issued a new regulation in March of 1983, the Ministry of the Environment failed, in our view, to impose adequate waste disposal procedures. We prescribe further improvements to the new regulation. We also recommend that steps be taken to identify disposal sites in which substantial quantities of asbestos have been deposited and to develop policies governing any redevelopment that might take place on such sites.

## **F. Compensating Victims: Asbestos and Its Implications**

To broach the subject of compensating the victims of industrial disease is to enter the realm of the Workers' Compensation Board (WCB). At the same time that we were inquiring into this agency's performance in the sphere of asbestos-related disease, it was the subject of another official study being conducted at the behest of the Minister of Labour by Professor Paul C. Weiler. We have endorsed, in whole or in part, a number of recommendations formulated by Professor Weiler. Given our sharply focused terms of reference, we have also probed deeply and prescribed specifically with respect to asbestos-related compensation.

No issues before this Commission proved more sensitive than those posed by workers' compensation. The Ontario WCB is a paradox. From one perspective, it is, in the sphere of asbestos disease, one of the most progressive compensation agencies in the world. This perspective is valid: the Ontario Board was the first workers' compensation agency in North America to compensate lung cancer in asbestos workers who did not also have clinical asbestosis; it has been the leader in compensating laryngeal and gastrointestinal cancer among asbestos workers; it has pioneered in rehabilitation and in outreach measures designed to identify workers whose disease arose from occupational exposure to asbestos. From the other perspective, the Board appears arbitrary and capricious, lacking in top-down direction and procedural fairness. Regrettably, we find this latter perspective eminently valid.

There is an explanation for the paradox of these conflicting and valid perceptions. Under its Act, the Board has been vested with broad discretion which it has used for enlightened ends. However, this same discretion, because it is inadequately structured and lacks confinement, is the breeding

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<sup>46</sup>For elaboration, see Chapter 11, Section D.

ground of arbitrariness.<sup>47</sup> Part of the problem lies with the Act rather than the Board. We, therefore, enthusiastically endorse Professor Weiler's recommendations that envisage a Workers' Compensation Appeals Tribunal, backed by Medical Review Panels, and issuing written, reasoned decisions. Like Professor Weiler, we urge that the Act be amended so that the Corporate Board will have a majority of outside, part-time directors, differing from him only in prescribing a somewhat larger membership.<sup>48</sup>

Although part of the Board's problem lies with the Act, part is self-inflicted. Specifically in the sphere of asbestos-related disease, the Board has failed to use a section of its Act which creates a statutory presumption in favour of claimants whose disease is directly associated with exposure to an industrial process.<sup>49</sup> Because we view mesothelioma and asbestosis as asbestos-specific diseases, we recommend that the link between these diseases and asbestos exposure be covered by statutory presumption, and that this presumption, now rebuttable, be made irrebuttable.<sup>50</sup>

While the Board deliberately chose, some thirty years ago, not to avail itself of the statutory presumption in its own Act, it nonetheless attempted to structure its discretion by developing industrial disease guidelines. Claimants who satisfied the terms of these guidelines would be automatically compensated, provided the facts of their cases were verified; claimants outside the guideline would be scrutinized on a case-by-case basis. We review the guideline-setting process, with specific reference to asbestos-related disease, and find that it has been informal, internal, unsystematic, and piecemeal and that it has done nothing to dispel perceptions of Board arbitrariness.<sup>51</sup> We recommend that the formulation of eligibility rules should henceforth involve an Advisory Council on Industrial Disease Policy (ACIDP), empowered to appoint expert panels to assist in the development of such rules. More generally, we prescribe that the terms of reference of the ACIDP, whose chairman should be a member of the Corporate Board, should embrace all matters of industrial disease policy and point out specifically the role this Council could play in reviewing rehabilitation programmes, monitoring and improving outreach measures, and considering appropriate methods of financing the cost of industrial disease compensation.<sup>52</sup>

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<sup>47</sup>This is a recurring theme throughout Chapters 12 and 13.

<sup>48</sup>For elaboration of our views on appeals and on the structure of the Corporate Board, see Chapter 12, Section C.1.

<sup>49</sup>For elaboration, see Chapter 12, Section B.2.

<sup>50</sup>For elaboration, see Chapter 12, Section C.2.

<sup>51</sup>Our grounds for this finding are set out in Chapter 12, Section B.3.

<sup>52</sup>The structure and role of the ACIDP are set out in Chapter 12, Section C.3; for yet further elaboration on its role see in Chapter 14, the concluding portions of each of Sections A, B, and C.



Among numerous recommendations addressing asbestos-related compensation matters, we highlight, for the purpose of this overview, those which focus upon the procedural and substantive issues posed by the compensation of asbestosis.<sup>53</sup> This irreversible and normally progressive condition, which can itself be fatal and in any event substantially reduces life expectancy, is a classic instance of an industrial disease whose victims are eligible for partial disability benefits. For decades, the Board has used a committee of experienced chest physicians, currently known as the Advisory Committee on Occupational Chest Diseases (AC OCD), to diagnose claimants and determine each patient's quantum (percentage) of medical impairment. The quantum of impairment is determined in accordance with the degree of physical impairment that medicine considers to be clinically measurable. We consider at length the fact that medicine has come to recognize the reality of psychological as well as physical impairment. We then review evidence, from both medical literature and sworn testimony, that the diagnosis of a condition such as asbestosis has adverse psychological consequences for the patient. We consider as well the fact that the courts, in calculating tort liability awards, have recognized psychological impairment notwithstanding the extreme difficulties that can be posed by its measurement. On the basis of all these considerations, we conclude that victims of asbestosis should be compensated for their combined physical and psychological impairment. By directive of the Corporate Board, the AC OCD should henceforth determine, by clinical diagnosis, the class of physical impairment to which each claimant should be assigned. Three classes, internationally recognized in the medical literature, should be used: Mild, Moderate, and Severe. The percentage of impairment recognized for compensation purposes should be stipulated by the Corporate Board's directive rather than determined by the AC OCD. This percentage should recognize the combined physical and psychological impairment of asbestosis victims in accordance with the class of physical impairment to which they have been assigned by the AC OCD and should be, where the AC OCD finds Mild impairment, 30%; where it finds Moderate impairment, 60%; and where it finds Severe impairment, 100%.<sup>54</sup> In addition to this substantive matter, we consider numerous procedural issues posed by the operation of the AC OCD and its relationship to the Board and make the prescriptions we deem appropriate.

More generally, we take cognizance of the fact that the Board, to its credit, has a benefit of doubt policy to guide its adjudication process, provide observations on the extent to which this policy has been neither uniformly followed nor uniformly communicated, and conclude that benefit of

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<sup>53</sup>For elaboration of all the points summarized in this paragraph, see Chapter 13, Section B.

<sup>54</sup>This recommendation has implications for the eligibility of the survivors of deceased asbestotics for death benefits; the matter of eligibility rules in this domain is addressed in Chapter 13, Section C.



doubt should be elevated to the level of the *Workers' Compensation Act*.<sup>55</sup> We address as well problems that beset the quality of Board communications with claimants and prescribe accordingly.<sup>56</sup>

In 1975, the Board launched a pioneering programme to rehabilitate employees whose past exposure to asbestos was deemed as warranting removal from further exposure.<sup>57</sup> We assess the factors that account for why this programme has been plagued by controversy and find that its deficiencies were indeed genuine, but that they are also understandable. However, we are distressed to find that removal and rehabilitation provisions contained in the Regulation Respecting Asbestos invite a repetition of these deficiencies, or worse, and we develop recommendations based on the lessons taught by the Board's Special Rehabilitation Assistance Programme.

The domain of outreach involves measures that seek to identify individuals whose disease or death was occasioned by industrial exposure and is therefore compensable by law.<sup>58</sup> We consider the evidence that asbestos-related deaths may be going uncompensated in Ontario and in this light find outreach measures to be necessary and important. We then review the Board's outreach measures and while we find them commendable, we conclude that such measures in future should be undertaken as a joint responsibility of the Board and of the Ministry of Labour.

Finally, we assess the extent to which a workers' compensation system can play a role in the prevention of industrial disease.<sup>59</sup> We find that the objective of prevention is closely associated with other major goals, notably equity among employers and justice. We consider the provisions of the *Workers' Compensation Act* and its regulations which, together with Board practices, govern the financing of compensation costs and the assessment of penalties. We find that the asbestos experience identifies severe deficiencies, whether from the standpoint of prevention, equity, or justice, and prescribe accordingly.

## G. Learning from the Asbestos Experience

This entire Report is an exercise in learning from the asbestos experience and discerning the lessons it teaches about health hazards and their regulation. We close the Report with some observations concerning what the asbestos experience teaches us about hazard identification and about the

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<sup>55</sup>For elaboration, see Chapter 13, Section D.

<sup>56</sup>See Chapter 13, Section E.

<sup>57</sup>For elaboration of the points summarized in this paragraph, see Chapter 14, Section A.

<sup>58</sup>For elaboration of the points summarized in this paragraph, see Chapter 14, Section B.

<sup>59</sup>For elaboration of the points summarized in this paragraph, see Chapter 14, Section C.

process which is currently in place for designating hazardous substances in Ontario workplaces.

The asbestos story demonstrates that the process of hazard identification can unravel slowly and that regulatory responses can lag behind the knowledge that slowly accumulates while a mounting toll of disease and death is borne by workers who are thereby cast in the role of human guinea pigs.<sup>60</sup> The alternative to this unacceptable state of affairs lies in swifter identification through new pre-screening techniques and swifter regulatory response. Fortunately, society now recognizes that factors in the human environment contribute importantly to premature death, that medical treatment is limited in what it can do for many of these conditions, and that prevention is the best approach for the control of many diseases. Recent though it is, society's appreciation of these facts is spawning more effective approaches to hazard identification and more timely and effective regulation. If the asbestos experience in the workplace offers stark testimony to the shortcomings of earlier societal views that were indifferent to disease prevention, it also speaks for the importance of being discriminating in the apprehension of hazards and in the formulation of regulatory responses. By the 1970s, society was sensitized to the contribution of the environment to disease and to the importance of prevention. However, as the enormity of the health disaster that was befalling asbestos workers came to be appreciated by the public in the 1970s, regulations were imposed and protective actions were taken which did not recognize that a substance that kills in the workplace may pose insignificant risks in the indoor or outdoor environment. This speaks clearly for an enhanced hazard identification and risk assessment capacity in government and prompts us to urge the creation, preferably on a national basis, of a hazard identification and risk assessment agency with sophisticated scientific capacity and open means of communicating with elected representatives, the media, and regulatory officials. We also consider the need for more co-ordinated communication of information by hazard-regulating agencies and prescribe accordingly.

A royal commission should probably be the last body to comment on any governmental process from the standpoint of the amount of time it consumes. We nonetheless comment on the process whereby the Ministry of Labour designates hazardous substances for regulatory purposes.<sup>61</sup> In the asbestos case, this process consumed more than two years. The open and participatory aspects of the designation process, which account in large part for its measured pace, are in our judgement entirely praiseworthy. Nonetheless, in order to provide workers with some additional protection during the long process of designation, we believe that the Ministry of Labour should

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<sup>60</sup>For elaboration of the points summarized in this paragraph, see Chapter 15, Sections B and C.

<sup>61</sup>For elaboration of the points summarized in this paragraph, see Chapter 15, Section D.

consider the advisability of a two-stage procedure whereby a preliminary regulation would be issued at the time notice of intent to designate is given and remain in place until the final regulation is promulgated.

Was this Royal Commission really necessary? We pose the question with which we opened this chapter because it is for readers of this Report to answer. A final observation is in order: this Royal Commission lasted almost four years and cost the Ontario taxpayers \$1.7 million.

# **Chapitre 1    Rapport de la Commission royale d'enquête sur l'amiante : Vue générale**

## **A. Encore une commission royale d'enquête**

Cette Commission royale d'enquête était-elle vraiment nécessaire?

Qu'elles le méritent ou non, les commissions royales d'enquête au Canada sont réputées des moyens lents, compliqués et coûteux pour étudier des questions plus ou moins importantes placées sous le signe de la controverse publique. Les sujets ainsi étudiés représentent une gamme de questions presque infinie : ils ont parfois été aussi spécifiques que les affaires d'une seule personne et aussi vastes que l'avenir économique du pays tout entier. Pour notre part, nous avons été nommés par le Gouvernement de l'Ontario pour nous pencher sur des questions de santé et de sécurité dans la province en ce qui concerne l'utilisation d'une seule substance : l'amiante. Depuis des années déjà, avant même que la Commission ne fût créée en avril 1980, on connaissait les effets ravageurs de l'amiante sur la santé. On n'avait guère besoin de nous pour confirmer cet état de chose.

Cette dure réalité a, à juste titre, provoqué l'appréhension générale. L'amiante est présent partout : il est dans l'air que nous respirons, il est dans l'eau que nous buvons. On l'utilise depuis des générations pour sa remarquable propriété de résister au feu et aux acides. Le Canada en est le plus grand producteur des pays occidentaux. Partout dans le monde, l'amiante a semé la maladie et la mort chez littéralement des milliers de personnes qui ont été exposées à ses fibres microscopiques dans l'exercice de leur profession : mineurs, ouvriers de moulin et de fabrique, poseurs d'isolant et travailleurs exerçant d'autres métiers. Il s'avère que ces personnes ont réellement sacrifié leur vie pour avoir de quoi vivre, ainsi que le



proclame un ouvrage bien connu à ce sujet, *Dying for a Living*.<sup>1</sup> Quelles sont les conséquences de perpétuer ou non l'utilisation de l'amiante dans l'industrie? Et d'un point de vue plus général, sait-on si cette substance mortelle pour les travailleurs de l'industrie menace les autres à leur insu dans les bureaux, les aéroports, les écoles?

Qu'elle soit nécessaire ou non, la Commission n'est pas passée inaperçue. Sa première audience publique, tenue en octobre 1980, a attiré quelque 400 personnes et ce nombre a presque été égalé lors de la deuxième séance publique six semaines plus tard. Ces séances avaient été annoncées par les avis publiés dans 81 quotidiens et hebdomadaires de tous les coins de l'Ontario. Il était mentionné dans les avis que l'on pouvait obtenir sur demande une brochure documentaire sur notre plan d'activités. Envoyée en outre sur notre initiative à des personnes susceptibles de s'intéresser à notre mandat, cette brochure contenait une carte-réponse permettant au destinataire de s'abonner aux communications postales de la Commission, ou de signifier son intention de présenter un mémoire oral ou écrit. Plus de 2 000 personnes et organisations se sont inscrites et se sont ainsi tenues au courant des dates de chaque audience et de nos travaux de recherche. Pendant la semaine du 16 au 20 février 1981, nous avons siégé matin, midi et soir pour entendre les exposés oraux à Toronto, dans une ambiance aussi libre de formalités que possible, compte tenu de la présence intermittente des aveuglants projecteurs de la télévision. Les exposés sont venus de personnes de tout état et de toute condition, de l'ouvrier gravement malade ou de la veuve solitaire jusqu'au chef de grande entreprise ou dirigeant d'organisation ouvrière ou gouvernementale. D'autres audiences de ce genre ont suivi, le 27 mars 1981 à Windsor et le 8 juin 1981 à Toronto. Mais les commissions royales d'enquête ne se contentent pas de tenir des audiences sans formalités, du moins pas en Ontario; cela explique sans doute pour une bonne part leur lenteur et leur lourdeur légendaires.

Suivant l'article 5(1) de la loi sur les enquêtes publiques,

La commission doit accorder, au cours de l'enquête, à quiconque justifie l'intérêt qui le lie directement à l'objet de l'enquête, l'occasion de témoigner et d'appeler des témoins pour les interroger ou les contre-interroger lui-même ou par l'entremise de son avocat à l'égard de témoignages pertinents.<sup>2</sup>

En octobre 1980, nous avons annoncé que les personnes intéressées à s'inscrire comme parties en règle suivant les dispositions prévues à l'article

<sup>1</sup> Lloyd Tataryn, *Dying for a Living* (Toronto, Deneau and Greenberg Publishers Ltd., 1979).

<sup>2</sup>Traduction non officielle; texte anglais dans les Lois refondues de l'Ontario de 1980, chap. 411.

5(1) devaient le faire avant le 31 janvier 1981 et nous avons procédé à l'inscription en règle des vingt parties mentionnées à l'appendice A, à la fin du présent rapport. Nous avons décidé en outre que l'une des parties, les Victimes de l'amiante de l'Ontario (*Asbestos Victims of Ontario*), devait recevoir une aide financière publique pour la défrayer de ses frais de participation. Les Victimes de l'amiante de l'Ontario regroupe d'anciens travailleurs de l'industrie de l'amiante, leurs conjoints ou leurs veuves.

Les mois qui suivirent, les représentants des parties en règle ont rencontré périodiquement notre avocat-conseil pour établir par consensus la liste des témoins que l'on inviterait à témoigner. Le taux de réponse à nos invitations s'est avéré à peine inférieur à 100 pour cent. Les noms des témoins figurent à l'appendice B. Ces 53 hommes et femmes sont venus non seulement de l'Ontario et d'autres endroits au Canada, mais aussi de la Suède, du Royaume-Uni et des États-Unis pour témoigner sous serment. La plupart ont été interrogés et contre-interrogés pendant une journée entière et quelquefois deux. Une moitié environ de nos témoins étaient des scientifiques, constituant un répertoire international d'éminents spécialistes des effets de l'amiante sur la santé. Pour recueillir les témoignages en bonne et due forme, il a fallu 53 jours d'audience répartis principalement sur les mois de l'été 1981 et 1982.

Pendant que nous menions nos audiences, nous avons cherché, soit personnellement, soit par l'entremise de notre personnel, à étendre davantage nos connaissances par des observations directes et des contacts sans caractère officiel. Cela comportait, à titre d'exemple, des visites à certaines mines d'amiante du Québec, à l'École de médecine du Mount Sinai à New York, à certains établissements manufacturiers en Ontario et à quelques projets où on éliminait l'amiante des bâtiments. Nous avons aussi créé un programme de recherche pour mieux comprendre les questions qui s'imposaient à nous. Ces recherches ont produit deux volumes de documentation générale et dix études spécialisées constituant chacune un volume en soi, dont on trouvera les titres à l'appendice F; nous avons diffusé ces ouvrages dès leur publication afin de les soumettre à examen et à critique. Ces études touchent à toute une gamme de sujets, partant des difficultés techniques posées par la mesure des niveaux de poussières d'amiante et aboutissant à l'indemnisation des travailleurs atteints de maladies associées à l'amiante en Ontario. Comme les études ont commencé de paraître en février 1982, l'information qu'elles révélaient et le contexte qu'elles fournissaient vinrent se greffer utilement à nos audiences subséquentes. Le dernier jour d'audience officielle fut le 24 août 1982. Après avoir donné le temps aux parties en règle d'examiner les témoignages, nous sommes retournés une dernière fois en salle d'audience, pour entendre leur exposé final, le 28 janvier 1983. À part cette occasion, nous nous sommes enfermés pour délibérer avec notre directeur de recherche, notre avocat-conseil et notre coordonnatrice administrative, dans l'intimité de nos 12 études de

recherche et de documentation générale, de nos 88 mémoires écrits et de nos 8 738 pages de communications orales et témoignages faits sous serment.

Ce rapport est le fruit de nos délibérations. Sa longueur et son style — genre littéraire que nous qualifions de «commissionnesque» — garantissent que fort peu de gens le liront de la première à la dernière page. Dans ce chapitre, nous essayerons de distiller nos constatations pour en livrer le strict essentiel. Les lecteurs qui — sous l'empire de la colère ou du ravissement, de la stupéfaction ou de la curiosité, ou pour quelque autre motif — désirent des explications n'ont qu'à se reporter aux notes de renvoi pour savoir dans quels chapitres ou parties de chapitres se trouvent l'information de base et le raisonnement que nous avons suivis. Comme nous avons été normatifs ainsi qu'analytiques, nous livrerons l'essentiel de nos 117 recommandations officielles dans le corps de ce chapitre.

## B. Effets de l'amiante sur la santé

Les résidents de l'Ontario ont plus de raisons que les autres de ressentir l'appréhension que l'amiante a suscitée à l'échelle internationale. L'Ontario est le théâtre de ce que nous décrivons dans ce rapport comme une catastrophe de premier ordre au plan mondial en ce qui concerne la santé des travailleurs : l'usine Johns-Manville à Scarborough, dans la Communauté urbaine de Toronto. De 1948 à 1980, cette usine a utilisé un mélange de deux types d'amiante, le chrysotile et la crocidolite, pour fabriquer des tuyaux d'amiante-ciment. À divers moments de son exploitation, l'usine a aussi utilisé du chrysotile seul pour fabriquer de la planche d'amiante-ciment; de même, elle a employé, pour faire des isolants, du chrysotile et une troisième espèce d'amianté, l'amosite.<sup>3</sup>

En 1983, le nombre de décès dûs à l'exposition aux fibres d'amiante dans cette usine s'élevait à 68, si l'on se fie au nombre de demandes d'indemnité accordées par la Commission des accidents du travail.<sup>4</sup> Cette seule usine, où le nombre d'emplois n'a jamais dépassé 714 par année, a déjà entraîné plus de décès imputables à des maladies professionnelles que l'ensemble de l'industrie minière en Ontario, qui emploie au-delà de 30 000 travailleurs par année, ne provoque d'accidents mortels pendant une période type de quatre ans.

<sup>3</sup>Pour une description des différents types d'amianté, voir chapitre 2, section B.

<sup>4</sup>Pour un exposé complet de l'expérience de la maladie et de la mort dans cette usine, voir chapitre 3, section A. Pour un compte rendu du coût de l'indemnisation accordée aux victimes de cette usine, voir chapitre 14, section C. Un effort spécial de réadaptation déployé pour les travailleurs de cette usine par la Commission des accidents du travail est examiné au chapitre 14, section A.



Le bilan des décès pour cette usine que l'on a fermée en 1980 offre un témoignage sévère quant à la nature de la maladie à longue période de latence.<sup>5</sup> Le nombre a progressé lentement et inexorablement parce qu'une période de quelque 10 à 30 ans, ou plus longue, sépare le décès de sa cause, qui remonte au début de l'exposition à l'amiante. La catastrophe a donc pris de l'ampleur avec le temps; ainsi, par exemple, entre août 1981 et août 1982, année centrale dans l'existence de la Commission, 5 anciens employés sont morts d'un mésothéliome, forme rare de cancer spécifiquement associée à l'exposition aux poussières d'amiante. On risque peu de se tromper, et cela est tragique, si l'on affirme que les ouvriers ayant déjà travaillé dans cette usine continueront de succomber aux méfaits de l'amiante et que la catastrophe n'est pas consommée.

Les décès associés à l'amiante surviennent par mésothéliome. Les décès par cancer du poumon, cancer gastro-intestinal et cancer du larynx peuvent avoir diverses causes, mais entre autres, l'amiante. Les décès peuvent survenir par amiantose; comme son nom l'indique, c'est une maladie strictement causée par l'amiante. C'est une variété de fibrose pulmonaire qui peut être mortelle en soi, mais elle agit plus communément en exposant les malades à des causes mortelles apparentées et, surtout, elle engendre un état d'infirmité irréversible et généralement évolutif. Au moment où l'usine Johns-Manville a cessé ses opérations en 1980, 113 de ses employés exposés à l'amiante avaient obtenu une pension d'invalidité partielle de la Commission des accidents du travail étant donné les divers degrés d'infirmité dont ils étaient affligés suite à l'amiantose.

La catastrophe dans cette usine se range parmi les pires incidents que l'on ait signalé dans la littérature internationale consacrée aux épidémies associées à l'amiante. Elle inscrit le nom de Scarborough sur une liste infamante où l'on retrouve Charleston, en Caroline du Sud; Rochdale, en Angleterre; et une poignée d'autres localités.<sup>6</sup> De tous les cas de décès et d'infirmité qui sont dûs à l'amiante et qui ont bénéficié d'indemnités de la part de la Commission des accidents du travail de l'Ontario, la moitié est attribuable à l'usine de Scarborough. Le reste résulte d'une exposition due à des situations professionnelles et des applications industrielles très diverses, comme, par exemple, la fabrication de masques à gaz pendant la guerre, l'usinage de freins et la construction de bateaux. En Ontario, les employeurs dont certains employés ont eu une maladie ou sont morts à cause de l'amiante se répartissent sur toutes les régions; à l'exclusion de

<sup>5</sup>Pour une description des maladies associées à l'amiante et du phénomène de latence prolongée, voir chapitre 2, section D. Pour un examen plus poussé de l'intervalle de temps entre l'exposition initiale à l'amiante et la manifestation clinique de la maladie, voir chapitre 5, section E.4.

<sup>6</sup>Pour une analyse comparative de la maladie et de la mortalité chez les travailleurs de l'amiante dans diverses usines dans le monde, voir chapitre 5, section B.



Johns-Manville, seulement deux d'entre eux ont donné lieu à plus de 5 prestations d'indemnités.<sup>7</sup>

Il y a de quoi s'inquiéter au sujet de l'amiante en Ontario. Mais au fond, «au fin fond», que pense la Commission de la nature des menaces que l'amiante fait peser sur la santé? Le «fin fond» de notre pensée peut tenir dans les énoncés suivants :

1. Les maladies associées à l'amiante présentent un cas classique d'un phénomène appelé la relation dose-réponse. Plus l'individu est exposé à l'amiante (la dose), plus il est probable qu'il soit atteint de maladie (la réponse). Inversement, moins l'individu est exposé à l'amiante, moins il est probable qu'il soit atteint de maladie.<sup>8</sup> Cependant, quand l'exposition se situe à de faibles concentrations, l'évaluation du risque de maladie s'embrouille dans l'incertitude. Cette incertitude naît de plusieurs facteurs, notamment la difficulté de réaliser les mesures, les lacunes de l'épidémiologie et le manque de données.<sup>9</sup> Dès lors, la prudence exige de présumer que toute exposition comporte un risque de maladie.

2. Presque tout le poids de la preuve scientifique penche du côté établissant que l'inhalation (la respiration) de fibres d'amiante aboutit à la maladie. Aucune preuve formelle n'existe à l'effet que la maladie peut résulter de l'ingestion (l'avalement) de fibres d'amiante et il existe seulement des hypothèses très spéculatives pour expliquer comment l'ingestion rendrait malade.<sup>10</sup>

3. Les fibres d'amiante qui présentent la plus forte probabilité de nocivité par inhalation sont longues et minces. La «longueur» et le «diamètre» sont évidemment d'ordre relatif : les fibres se mesurent en microns et le micron est égal à un millionième de mètre. Les fibres d'amiante sont dangereuses quand elles dépassent 5 microns de longueur, ou peut-être 8, et que leur diamètre est inférieur à 1,5 micron, ou peut-être à 0,25 micron.<sup>11</sup>

4. Il y a de solides témoignages voulant que les fibres de crocidolite et d'amosite seraient plus nocives que les fibres de chrysotile, surtout parce qu'elles ont tendance à correspondre en longueur et en diamètre à la

<sup>7</sup>Pour un exposé du volume des prestations accordées pour l'indemnisation de maladies et de décès chez divers employeurs en Ontario, voir chapitre 3, section A.

<sup>8</sup>Pour un examen des relations dose-réponse en ce que concerne les maladies associées à l'amiante, voir chapitre 5, section E et chapitre 7, section C.

<sup>9</sup>Pour un examen de ces facteurs, voir chapitre 4, section D.

<sup>10</sup>Pour un exposé de la preuve biologique quant à l'ingestion des fibres d'amiante, voir chapitre 4, section B.2. Pour un examen de la preuve épidémiologique, voir chapitre 5, section I.

<sup>11</sup>Pour une analyse de l'importance des dimensions de la fibre, voir chapitre 4, section B et chapitre 5, section D.

catégorie la plus dangereuse, mais aussi parce qu'elles ont une très grande aptitude à flotter dans l'air et, de là, à être inhalées par respiration. Il est également possible que la crocidolite et l'amosite soient plus nocives à cause de leur composition chimique, mais cette possibilité relève du domaine de la conjecture.<sup>12</sup>

5. Toutes les espèces de fibres peuvent causer n'importe quelle maladie associée à l'amiante, mais le mésothéliome est le résultat le plus probable d'une exposition à la crocidolite, quoiqu'il soit souvent l'aboutissement d'une exposition à l'amosite; toutefois, il n'est que faiblement associé à une exposition au chrysotile.<sup>13</sup>

6. Quel que soit le type d'amiante, la probabilité d'en respirer les fibres de dimensions particulièrement dangereuses dépend beaucoup du procédé d'utilisation de l'amiante. Ainsi, par exemple, la fabrication de garnitures de frein, qui comporte des opérations de forage et de polissage, risque beaucoup moins de produire des fibres nuisibles que la fabrication textile, qui comporte des opérations de filage et de tissage.<sup>14</sup>

7. Les victimes des maladies spécifiques dues à l'amiante ont été presque invariablement exposées à l'amiante à cause de leur travail. Cependant, il est indéniable que le mésothéliome atteint des personnes qui ont partagé le logis d'un travailleur à l'époque où celui-ci était exposé aux poussières d'amiante. Il est peu plausible que des personnes aient contracté une maladie parce qu'elles étaient exposées au voisinage d'une installation industrielle utilisant de l'amiante.<sup>15</sup> Nous n'avons pas trouvé de preuve à l'effet que la maladie atteint des personnes qui respirent de l'amiante à l'extérieur, en plein air, ou l'inhalent parce qu'elles occupent des bâtiments où se trouve de l'amiante.<sup>16</sup>

8. L'amiantose est une maladie attribuable uniquement à l'exposition professionnelle à l'amiante. Il est fort évident qu'à un très bas niveau d'exposition à l'amiante, la fibrose pulmonaire, si effectivement elle peut être déclenchée, ne se développera pas jusqu'à produire une amiantose qualifiée

<sup>12</sup>Pour un examen de la preuve qui indique que la crocidolite et l'amosite ont tendance à être plus nocives que le chrysotile, voir chapitre 5, section C. Pour un exposé de l'importance des dimensions de la fibre et de l'importance possible de la nature chimique de la fibre, voir chapitre 5, section D.

<sup>13</sup>Pour un exposé et une analyse de la preuve concernant l'incidence du mésothéliome en rapport avec les différents types de fibre, voir chapitre 5, sections C.4, C.6 et D; et chapitre 7, sections C et F.

<sup>14</sup>Pour un compte rendu de l'état de santé des travailleurs employés dans divers procédés d'utilisation de l'amiante, voir chapitre 5, section B et chapitre 7, section C. Pour un examen des effets du procédé sur les dimensions de la fibre et des effets qui en découlent pour la santé, voir chapitre 5, section D et chapitre 7, sections C et F. Pour une description des différents procédés industriels, voir chapitre 6, section C.

<sup>15</sup>Pour plus d'explications sur ces points, voir chapitre 5, section E.

<sup>16</sup>Voir chapitre 11, section C et chapitre 9.

par diagnostic clinique ou une manifestation quelconque qui puisse même provoquer le moindre malaise.<sup>17</sup>

9. Il n'y a aucun lien entre l'usage du tabac et le mésothéliome. L'amiantose ne peut pas être causée par le tabac, mais il est possible que le tabagisme accélère le développement du fibrome. Enfin, bien que l'amiantose puisse causer à elle seule le cancer du poumon, il est nettement prouvé que l'amiante et le tabac se conjuguent avec une telle puissance synergique pour engendrer cette maladie que fumer et travailler dans l'amiante équivaut à jeter de l'huile sur le feu.<sup>18</sup>

Nous avons fait plusieurs autres constatations quant aux effets de l'amiante sur la santé, mais ce qui précède se ramène aux faits saillants. Qu'est-ce que ces constatations comportent? Nous répondrons à la question en examinant d'abord l'amiante dans l'industrie fixe, ensuite l'amiante dans les bâtiments et, enfin, l'amiante dans d'autres contextes.

## C. L'amiante dans les lieux de travail fixes

Les lieux de travail fixes, en l'occurrence les mines et les usines de fabrication, constituent une compartimentation des activités reliées à l'amiante dans un but de réglementation, et cela pour diverses raisons. Premièrement, ces activités sont facultatives; elles pourraient être interdites si l'on jugeait qu'elles comportent des risques inacceptables. Deuxièmement, ces activités, du fait qu'elles sont circonscrites dans des lieux de travail fixes, se prêtent à des techniques de travail et à des équipements de contrôle qui réduisent la concentration des fibres d'amiante dans le milieu auquel les travailleurs sont exposés. Troisièmement, encore une fois parce que le travail s'effectue dans un lieu fixe, il est possible de surveiller de façon systématique la concentration de fibres d'amiante dans l'air. La méthode de mesure usuelle comprend : (i) le prélèvement d'un échantillon d'air par ce que l'on désigne comme la méthode de la membrane filtrante; (ii) le dénombrement des fibres captées dans chaque échantillon d'air à l'aide d'un microscope optique; et (iii) l'expression du nombre de fibres décelées en terme de tant de fibres par centimètre cube d'air (f/cc).<sup>19</sup>

Suivant les dispositions prévues par la *Loi sur la santé et la sécurité au travail*, l'amiante est une substance désignée et assujettie au règlement concernant l'amiante. Ce règlement enjoint les exploitants d'installations fixes de prendre toutes les mesures et dispositions nécessaires pour garantir

<sup>17</sup>Pour un examen de cette affirmation, voir chapitre 5, section E.3.

<sup>18</sup>Pour un examen de la relation entre le tabagisme, l'exposition à l'amiante et la maladie, voir chapitre 5, section F.

<sup>19</sup>Pour une description détaillée et une analyse de la méthode de la membrane filtrante, voir chapitre 7, section B.



que l'exposition des travailleurs aux poussières d'amiante soit réduite au plus bas niveau pratique et il stipule les normes à ne pas dépasser. Ces normes se situent à 1 f/cc pour le chrysotile, à 0,5 f/cc pour l'amosite et à 0,2 f/cc pour la crocidolite. Le règlement s'étend à tous les types d'installations fixes dans l'industrie utilisatrice d'amiante et n'établit aucune distinction entre les activités industrielles.

L'extraction minière de l'amiante et son utilisation dans toutes sortes de procédés de fabrication font partie de l'histoire de l'Ontario. L'exploitation minière, qui comporte l'extraction de l'amiante chrysotile, est à présent une activité marginale quasi inexistante; à notre connaissance, l'industrie manufacturière se limite à l'heure actuelle à l'emploi de l'amiante chrysotile et l'utilise surtout pour fabriquer des produits de friction, notamment les freins d'automobile.<sup>20</sup>

À quel risque s'exposent les travailleurs dans ces différents contextes et, quant à cela, dans toute autre activité impliquant l'utilisation de l'amiante en milieu industriel fixe? De tous les agents carcinogènes d'utilisation industrielle dans le monde, l'amiante est de ceux qui ont fait l'objet d'études les plus poussées. Il en résulte une base de données qui permet d'estimer le risque de maladie selon le niveau d'exposition professionnelle. Nous nous sommes servi de modèles d'incidence pathologique et de données tirées de maintes études épidémiologiques concernant des travailleurs exposés à l'amiante pour faire une projection du risque éventuel de maladie que l'exposition à tout type de poussière d'amiante associée à différents procédés de fabrication peut entraîner à divers degrés de concentration.<sup>21</sup>

En nous basant sur cette analyse, nous concluons qu'il nous est impossible de fermer les yeux sur toute activité de fabrication qui exige l'emploi de l'amiante crocidolite ou amosite.<sup>22</sup> Il devrait être interdit indéfiniment d'utiliser ces deux types d'amiante en Ontario.<sup>23</sup> En ce qui con-

<sup>20</sup>Pour une description de l'exploitation minière de l'amiante en Ontario, voir chapitre 6, section F; l'activité manufacturière est décrite au chapitre 6, section B.

<sup>21</sup>Ces estimations sont détaillées au chapitre 7, section C. Une description technique du modèle d'évaluation du risque figure à l'appendice du chapitre 7.

<sup>22</sup>Pour les critères que nous avons élaborés en vue d'évaluer quand une substance doit être soumise à une norme, quand elle doit être interdite, et la relation entre une substance donnée et les substituts possibles, voir chapitre 7, section E. La faisabilité et le coût des méthodes de contrôle de l'amiante sont examinés au chapitre 6, section C. Pour un examen des substituts et de leurs effets sur la santé, voir chapitre 6, sections D et E.

<sup>23</sup>Voir chapitre 7, section F.3. L'interdiction que nous recommandons prend la forme d'une prohibition absolue par le ministère du Travail, laquelle pourrait être abolie seulement si les risques pour la santé étaient considérablement réduits. Dans le cas de l'amosite, il faudrait fixer l'exposition maximum à 0,1 f/cc, soit un niveau inaccessible à l'heure actuelle (voir recommandation 7.14); dans le cas de la crocidolite, l'exposition maximum, si même l'on peut y songer, devrait être fixée à 0,02 f/cc (voir recommandations 7.12 et 7.13).



cerne le chrysotile, il faut distinguer les procédés industriels qui utilisent ce type d'amiant. La norme que le règlement concernant l'amiant impose à l'heure actuelle pour tous les procédés comportant du chrysotile est de 1 f/cc. Si cette norme est rigoureusement observée de sorte que, au niveau d'une moyenne pondérée sur 8 heures, elle dépasse rarement ce plafond, nous estimons que la moyenne du niveau d'exposition chez les travailleurs n'est pas supérieure à 0,5 f/cc.<sup>24</sup> Notre base de données nous permet de calculer le risque de maladie chez les travailleurs employés dans les milieux industriels suivants : mines et moulins; fabrication générale, sauf des produits textiles et des ciments; fabrication textile; fabrication de produits du ciment. Dans le cas de l'extraction minière et du traitement au moulin du chrysotile et dans le cas de la fabrication générale utilisant le chrysotile, nous constatons que le risque de maladie associé à l'exposition au chrysotile dans une limite normative de 1 f/cc, dûment appliquée, donne par projection un taux de mortalité bien inférieur au taux de mortalité résultant des accidents de travail dans l'ensemble de l'industrie manufacturière en Ontario. Par conséquent, ce risque se situe tout à fait dans les limites acceptables par la société en fait de risques professionnels.<sup>25</sup>

La fabrication de textiles avec du chrysotile est un cas entièrement différent. En-deçà de la limite de 1 f/cc dûment respectée, le risque de mortalité associé à cette transformation de l'amiant est estimé supérieur à la mortalité due aux accidents dans l'industrie de fabrication et il approche ou dépasse la mortalité d'origine accidentelle dans tout le secteur minier en Ontario. Au regard des accidents de travail, cette activité est depuis longtemps parmi les plus dangereuses de toutes en Ontario. Pour que le secteur de la fabrication de produits textiles comportant du chrysotile puisse réduire le risque de décès dû à la maladie au niveau qui prévaut dans les autres secteurs d'utilisation du chrysotile, il faudrait fixer la norme à 0,04 f/cc, c'est-à-dire à un niveau beaucoup trop bas pour que les techniques de mesure actuelles puissent en assurer l'application. Nous constatons par conséquent que ce procédé d'utilisation du chrysotile ne peut pas être toléré en Ontario et qu'il doit être interdit aussi longtemps qu'il ne sera pas possible de convaincre le ministère du Travail que sa pratique ne comporterait pas de risques plus grands que ceux posés par les mines et la fabrication générale utilisant le chrysotile.

Le risque que nous associons à la manipulation du chrysotile dans les mines et la fabrication générale peut être moindre que le risque d'accident dans la fabrication, mais il n'en demeure pas moins important; il constitue

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<sup>24</sup>Pour la base de calcul, ainsi qu'un examen approfondi des erreurs d'échantillonnage et de mesure, de la fréquence de l'échantillonnage et autres questions du même ordre, voir chapitre 7, section B.

<sup>25</sup>Pour un examen approfondi de ce point et des points résumés dans le reste de ce paragraphe et dans le paragraphe suivant, voir chapitre 7, section F.

en effet un risque de maladie chez le travailleur de l'amiante qui, comme tout employé d'usine, s'expose à des accidents de travail mortels. Nous considérons donc ce qui suit comme essentiel pour que l'on tolère l'utilisation du chrysotile à une limite normative de 1 f/cc : (i) Tout travailleur doit être assidûment informé du risque qu'il court, ainsi que le prévoit le règlement concernant l'amiante. (ii) Le règlement doit être rigoureusement appliqué afin que la norme de 1 f/cc garantisse en réalité que l'exposition moyenne réelle soit de 0,5 f/cc. (iii) Le règlement, la *Loi sur la santé et la sécurité au travail*, ainsi que les pratiques et l'organisation du ministère du Travail doivent être modifiés afin que le gouvernement, l'assemblée législative, le public et, par-dessus tout, les travailleurs concernés soient assurés que le règlement touchant l'amiante chrysotile est appliqué efficacement.<sup>26</sup>

À cet égard, nous avons fait des recommandations expresses qui vont de la mise au point de méthodes plus exactes pour l'échantillonnage de l'air jusqu'à la participation accrue du travailleur au processus d'application et de surveillance. Nous signalons que l'une de nos recommandations propose la création d'une unité de surveillance des substances désignées, relevant du ministère du Travail, afin de renforcer son rôle d'inspection dans les cas où le lieu de travail abrite des substances désignées. À part son directeur, qui devrait être formé aux techniques d'enquête, l'unité n'exigerait pas du ministère d'embaucher du personnel nouveau, puisqu'elle ferait plutôt appel à des équipes interdisciplinaires composées de professionnels qualifiés en science et technologie à l'emploi de la Direction de la santé au travail; le directeur de l'unité assemblerait l'équipe voulue pour visiter sans préavis les usines utilisant des substances désignées.<sup>27</sup>

## D. L'amiante dans les bâtiments

En Ontario, comme ailleurs, l'amiante a été utilisé couramment jusqu'en 1973 comme isolant soufflable ignifuge et insonorisant, et comme revêtement calorifuge pour les tuyaux et les chaudières de chauffage.<sup>28</sup> L'amiante est donc présent dans un grand nombre de bâtiments à multiples étages, y compris les immeubles à bureaux, les écoles, les usines et les aéroports. Le type d'amiante qui s'y trouve généralement est non seulement du chrysotile, mais aussi de l'amosite.

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<sup>26</sup>Pour un examen approfondi du règlement concernant l'amiante, le système de responsabilité interne et l'application, voir chapitre 8.

<sup>27</sup>Voir chapitre 8, section E.1.

<sup>28</sup>Pour un examen de l'application et des caractéristiques de l'isolant soufflable et de l'isolant pour tuyaux et chaudières de chauffage, des bâtiments où on en trouve et des types d'amiante utilisés, voir chapitre 9, section B.

Naturellement, la présence d'amiante chrysotile et d'amiante amosite dans les bâtiments a suscité une vive inquiétude chez le public. Cette appréhension est-elle justifiée? À cette question, nous répondrons en deux temps. Premièrement, l'amiante peut en effet poser un danger sérieux pour les ouvriers qui effectuent des travaux tels que l'enlèvement de l'amiante et la démolition de bâtiments, ou qui exécutent toutes sortes de travaux d'entretien des bâtiments.<sup>29</sup> Deuxièmement, l'inhalation de fibres de chrysotile et d'amosite par les occupants d'immeubles ne pose presque jamais de problème de santé, sauf peut-être quand on remue l'amiante et qu'on s'expose alors à un niveau particulièrement élevé de poussières, surtout lors des opérations d'enlèvement de l'amiante.<sup>30</sup>

Passons tout de suite à un examen plus approfondi de notre deuxième observation. Elle suit pour l'essentiel la ligne de pensée tracée par le comité consultatif du Royaume-Uni sur l'amiante (*United Kingdom Advisory Committee on Asbestos*) en 1979. Malgré cette constatation faite par les Britanniques, il semble que l'inquiétude publique quant au risque que l'amiante fait courir aux occupants d'un bâtiment ne veuille pas s'apaiser. Le cas échéant, c'est un argument de poids pour que les organismes publics agencent et coordonnent mieux leurs communications. Nous remarquons en particulier que l'inquiétude du public est attribuable en grande partie aux organismes publics chargés de contrôler l'usage de l'amiante, car ces organismes négligent de tirer au clair le facteur primordial qui domine les mesures du niveau d'amiante dans l'air des bâtiments. L'Ontario offre l'exemple parfait d'un domaine de compétence où deux organismes différents ont agi sans aucun souci de cohésion dans leurs communications. Le ministère du Travail de l'Ontario a promulgué à grand renfort publicitaire les normes obligatoires dans les lieux de travail fixes. Nous l'avons vu, la norme pour le chrysotile est de 1 f/cc. Parallèlement, le ministère de l'Environnement de l'Ontario a pour norme 0,04 f/cc en ce qui concerne la présence d'amiante dans l'air ambiant (habituellement assimilé à l'air dans les bâtiments). À première vue, il semble que la norme du ministère de l'Environnement est 1/25 de celle fixée par le ministère du Travail. Mais cela n'est pas vrai. La norme de 1 f/cc du ministère du Travail s'applique à un contexte où le dénombrement des fibres contenues dans l'échantillon d'air est effectué à l'aide d'un microscope optique. Par contre, la faible concentration de fibres envisagée par le ministère de l'Environnement s'appuie sur un dénombrement des fibres à l'aide d'un microscope électronique.<sup>31</sup> Le microscope électronique détecte des quantités de fibres invisibles au microscope optique. Ainsi, le comptage électronique des fibres

<sup>29</sup>Pour un examen des expositions à l'amiante que les travailleurs s'occupant des bâtiments ont encourues ou peuvent encourir, voir chapitre 9, sections B et D.

<sup>30</sup>Pour une analyse des expositions à l'amiante chez les occupants d'immeubles, voir chapitre 9, section D.

<sup>31</sup>Pour un examen de la norme concernant l'air ambiant, voir chapitre 11, sections C.4 et C.5.



d'amiante présentes dans l'air des bâtiments aboutira à un nombre plus élevé, l'équivalent d'un ordre de magnitude ou environ 10 fois plus de fibres que par comptage optique.<sup>32</sup> Il s'ensuit que la norme de 0,04 f/cc du ministère de l'Environnement se rapproche beaucoup moins de 1/25 et beaucoup plus de 1/250 de la norme établie au ministère du Travail.

Nous constatons qu'en plein air les concentrations de fibres en suspension sont souvent inférieures à 0,001 f/cc selon des mesures effectuées par microscope électronique, et qu'elles n'atteignent presque jamais 0,01 f/cc.<sup>33</sup> Par comptage optique, ces concentrations se situeraient dans l'ordre de magnitude inférieur. Chose sans doute plus importante, nous concluons que l'air dans les bâtiments dotés d'une isolation thermique à l'amiante soufflé atteint habituellement une moyenne inférieure à ce qui équivaut à 0,001 f/cc par comptage optique et touche des sommets rarement supérieurs à 0,01 f/cc. Supposons que l'air d'un bâtiment est contaminé par le chrysotile et par les fibres plus nocives de l'amosite, et faisons le calcul estimatif du risque que courent les occupants quand ce mélange de fibres atteint 0,001 f/cc. Le risque de décès consécutif à une maladie due à l'amiante chez une personne qui aurait occupé l'immeuble pendant 10 ans est 50 fois moindre que le risque de décès accidentel que cette personne courrait en voyageant 10 milles par jour en auto pour se rendre à cet immeuble matin et soir pendant la même période. À ce niveau, nous considérons négligeable le risque auquel les occupants d'un immeuble sont exposés et nous trouvons par conséquent que l'amiante présent dans l'air d'un bâtiment ne menacera presque jamais la santé des occupants.<sup>34</sup>

On ne peut toutefois pas affirmer la même chose à l'égard des travailleurs employés à des travaux de démolition, d'enlèvement de l'amiante ou d'entretien de bâtiments. Ces travailleurs peuvent être exposés aux fibres d'amiante à des niveaux qui dépassent de beaucoup la norme appliquée dans les installations industrielles fixes.<sup>35</sup> Le travailleur est ici exposé à l'amiante amosite et à l'amiante chrysotile. L'exposition, en ce cas-ci, peut se produire par vagues brèves mais intenses, capables de vaincre les défenses normales du poumon humain. Quand il s'agit de démolition, d'enlèvement et de certains travaux de réparation, le lieu de travail n'est pas fixe et s'apparente plutôt à un chantier de construction typique. Il s'ensuit que la protection du travailleur peut mieux se réaliser en précisant les méthodes de travail qu'en contrôlant la qualité de l'air dans le but de

<sup>32</sup>Pour un examen des rapports entre le comptage optique des fibres et le comptage des fibres par microscope électronique, voir chapitre 7, section B et chapitre 11, section C.1.

<sup>33</sup>Pour un examen des données concernant les concentrations d'amiante à l'extérieur, voir chapitre 11, section C.2.

<sup>34</sup>Pour une analyse du risque pour la santé que l'exposition à l'amiante pose pour les occupants d'immeubles, voir chapitre 9, section E.

<sup>35</sup>Pour des données sur l'exposition à l'amiante chez les travailleurs s'occupant des bâtiments, voir chapitre 9, sections B et D et chapitre 10, section C.



satisfaire à des normes, et nous donnons quantité de conseils à ce sujet.<sup>36</sup> Pour la démolition des bâtiments, le risque potentiel d'exposition est d'une telle magnitude que nous prescrivons expressément d'enlever tout l'amiante friable des bâtiments avant de les démolir.<sup>37</sup>

Depuis 1979, de nombreux conseils scolaires en Ontario, bénéficiant d'une subvention de quelque 26 millions de dollars du gouvernement provincial, ont déployé énormément d'efforts pour éliminer l'amiante des écoles.<sup>38</sup> Cela répondait directement aux pressions des parents inquiets pour la santé de leurs enfants. Il y a de solides arguments à l'effet que la possibilité d'apparition d'un mésothéliome est en rapport avec la période de temps écoulée depuis l'exposition initiale, ce qui signifie que plus une personne est jeune lorsqu'elle est exposée à l'amiante pour la première fois, plus elle risque d'avoir un mésothéliome au cours de sa vie.<sup>39</sup> Alors même que ce fait est reconnu et que par surcroît nous tenons compte de l'hypothèse selon laquelle les enfants seraient plus vulnérables aux maladies dues à l'amiante,<sup>40</sup> le danger pour la santé des enfants demeure négligeable, étant donné que l'exposition à l'amiante dans les écoles se situe habituellement à un niveau extrêmement bas. Les exceptions se rencontrent quand des morceaux d'isolant d'amiante sont remués ou lorsque ceux-ci se détachent du plafond. Il en résulte que, sauf exceptions, l'élimination de l'amiante de toutes les écoles n'était pas justifiée au regard du risque pour la santé des élèves. Dans quelques cas, il fallait sans doute contrôler l'amiante afin de protéger les travailleurs contre l'exposition à l'amiante si leurs tâches de rénovation, d'entretien ou de ménage les obligeaient à remuer de l'isolant d'amiante. Mais le risque que la plupart des écoles contenant de l'amiante posaient aux occupants ou aux travailleurs ne justifiait nullement une initiative à pareille échelle et allure. Si résultat il y a, cette initiative *a accru* sensiblement, par son étendue et son allure, le danger pour les travailleurs exécutant directement des opérations de contrôle. Les opérations d'urgence sont invariablement synonymes de l'entrée dans un domaine spécialisé d'entrepreneurs et de travailleurs inexpérimentés. Plusieurs opérations visant à contrôler l'amiante dans les écoles ont été effectuées d'une manière qui, à en juger par les témoignages devant nous, peut avoir exposé les travailleurs concernés à un risque important. Cependant, il ne fait aucun doute que l'urgence de l'opération visant à contrôler l'amiante dans les

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<sup>36</sup>Pour une explication sur la réglementation visant les méthodes au lieu d'une réglementation établissant des normes, voir chapitre 10, section A.

<sup>37</sup>Voir chapitre 10, section C.3. Pour une analyse qui compare le coût de l'enlèvement de l'amiante pendant que le bâtiment est encore utilisé au coût de l'enlèvement de l'amiante juste avant la démolition, voir chapitre 9, section E.

<sup>38</sup>Pour un examen du programme de contrôle de l'amiante dans les écoles de l'Ontario, voir chapitre 9, section E.

<sup>39</sup>Pour un examen de l'hypothèse selon laquelle le mésothéliome est fonction du temps, voir chapitre 5, section E.4.

<sup>40</sup>Voir chapitre 5, sections E.4 et H.

écoles est née d'un climat d'inquiétude publique; ainsi les conseils scolaires et le ministère de l'Éducation, tout comme leurs homologues ailleurs, ont agi de toute évidence en réponse à la demande du public.

## E. L'amiante dans d'autres contextes

Nous sommes tous exposés à l'amiante parce qu'on en trouve dans l'eau que nous buvons et dans l'air que nous respirons. On a compté jusqu'à 4 millions de fibres par litre d'eau potable dans des municipalités du Sud de l'Ontario (p. ex., Sarnia et la Communauté urbaine de Toronto) et jusqu'à 22 millions de fibres par litre dans des municipalités du Nord de l'Ontario (Thunder Bay). Les fibres sont presque toujours extrêmement courtes, c'est-à-dire inférieures à 1 micron de longueur.<sup>41</sup> Étant donné que ces fibres sont de petites dimensions et comme nous avons constaté que l'amiante provoque la maladie par inhalation et non par ingestion, nous arrivons à la conclusion que la présence de l'amiante dans l'eau potable ne met pas la santé en danger.<sup>42</sup> La même conclusion vaut pour l'amiante dans les aliments et les boissons,<sup>43</sup> et nous recommandons en effet que la Régie des alcools de l'Ontario lève l'interdiction frappant l'utilisation de filtres d'amiante dans la fabrication de la bière, du vin et des spiritueux. (Les consommateurs d'alcool en Ontario qui restent inquiets à ce sujet pourront noter que cette interdiction n'a jamais été réellement appliquée pour les boissons alcooliques importées.)

Nous avons examiné la question de l'amiante en plein air et nous avons noté la prévalence de fibres très courtes et de quantités peu élevées (un maximum de 0,0084 f/cc de toutes longueurs), même dans le voisinage d'un rampe d'autoroute à Toronto, endroit où l'on pourrait s'attendre à ce que le freinage des véhicules libère des fibres.<sup>44</sup> Nous concluons qu'un objectif visant la qualité de l'air ambiant, telle la norme de 0,04 f/cc adoptée par le ministère de l'Environnement, demeure important, non pas tellement parce qu'il y aurait du danger pour la santé à des niveaux d'exposition semblables, mais plutôt parce qu'on peut alors signaler tout écart inhabituel du niveau de pollution et en chercher l'origine. Quant à savoir si la norme de 0,04 f/cc est adéquate, nous recommandons que le ministère de l'Environnement la maintienne jusqu'à ce que la communauté internationale

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<sup>41</sup>Pour une description des niveaux et des dimensions des fibres d'amiante dans l'eau potable, voir chapitre 11, sections B.1 et B.2.

<sup>42</sup>Pour une analyse des effets sur la santé dus à l'ingestion de fibres d'amiante, voir chapitre 5, section I.

<sup>43</sup>Pour un examen de la présence de l'amiante dans les aliments et les boissons, voir chapitre 11, section B.3; la question de la présence de l'amiante dans les médicaments est examinée au chapitre 11, section B.4.

<sup>44</sup>Pour une description des niveaux d'amiante dans l'air extérieur, et pour plus d'explications sur les points résumés dans le reste de ce paragraphe, voir chapitre 11, section C.

reconnaisse des méthodes universelles pour mesurer l'air extérieur et pour permettre de discerner le sens des relevés numériques.

En ce qui concerne les biens de consommation contenant de l'amiante, nous nous inquiétons passablement de l'état de la réglementation.<sup>45</sup> La réglementation est, en un mot, désordonnée. Ainsi, par exemple, le ministère de la Santé et du Bien-être social du Canada a préconisé de cesser d'utiliser l'amiante dans la fabrication des sècheurs à cheveux portatifs, bien que les tests indiquent un niveau de fibres libérées équivalent aux niveaux normalement présents dans l'air extérieur. Pendant ce temps, l'amiante à l'état libre, qui peut occasionner la libération d'importantes quantités de fibres, n'est soumis à aucun règlement et se vend à l'heure actuelle dans les quincailleries partout au pays. Comme dans tant d'autres domaines de la vie au Canada, les biens de consommation sont sujets à une réglementation relevant de deux paliers de compétence, le gouvernement fédéral et le gouvernement provincial. Nous préconisons que le Gouvernement de l'Ontario prennent des dispositions, de préférence par voie de collaboration fédérale-provinciale, pour classer les biens de consommation en trois catégories. La première viserait les biens qui, à l'usage normal, seraient susceptibles de libérer des niveaux importants de fibres d'amiante (p. ex., l'isolant composé d'amiante libre); la deuxième comprendrait les objets qui libèrent des fibres quand on les coupe ou les sable, ou lorsqu'ils se dégradent (p. ex., panneau d'amiante-ciment ou gants d'amiante); la troisième catégorie regrouperait les produits dans lesquels l'amiante est incorporé ou enveloppé hermétiquement (p. ex., la plupart des appareils ménagers isolés à l'amiante). La vente des objets de la première catégorie devrait être prohibée (à moins qu'il n'existe pas de produit de remplacement adéquat ou que le produit soit destiné exclusivement aux entreprises manufacturières assujetties au règlement concernant l'amiante). Les objets de la deuxième catégorie devraient porter une étiquette et être accompagnés d'instructions pour en faire un usage sans danger. Les produits de la troisième catégorie ne seraient assujettis à aucun règlement.

Enfin, nous rangeons au nombre de nos préoccupations l'élimination des déchets d'amiante, non pas tellement parce qu'il en résulterait une pollution atmosphérique, mais plutôt parce qu'il faut protéger les travailleurs des services d'élimination des déchets contre les situations qui menaceraient leur santé.<sup>46</sup> Avant l'adoption d'un nouveau règlement en mars 1983, le ministère de l'Environnement a négligé, à notre avis, d'imposer des mesures appropriées pour l'élimination des déchets. Nous recommandons d'améliorer encore le nouveau règlement. En outre, nous préconisons de prendre des dispositions pour identifier l'emplacement des

<sup>45</sup>Pour plus d'explications à ce sujet et au sujet des points résumés dans le reste de ce paragraphe, voir chapitre 11, section A.

<sup>46</sup>Pour plus d'explications, voir chapitre 11, section D.



dépotoirs où l'on a déposé des déchets d'amiante en quantité considérable et d'adopter des politiques régissant tout réaménagement que l'on pourrait projeter pour ces espaces.

## **F. L'indemnisation des victimes : l'amiante et ses répercussions**

En abordant la question de l'indemnisation des victimes de maladies professionnelles, on pénètre sur le territoire de la Commission des accidents du travail (CAT). Pendant que nous enquêtons sur les réalisations de cet organisme en ce qui concerne les maladies associées à l'amiante, une autre étude officielle, commandée par le ministre du Travail et confiée au professeur Paul C. Weiler, s'intéressait au même organisme. Nous avons en-dossé, intégralement ou en partie, plusieurs recommandations formulées par le professeur Weiler. Comme notre mandat couvrait un champ bien restreint, nous avons exploré en profondeur et nous avons fait des recommandations spécifiques quant aux actes de compensation se rapportant à l'amiante.

Nulle autre question abordée devant notre Commission ne s'est avérée aussi délicate que celle de l'indemnisation des travailleurs. La CAT de l'Ontario est un paradoxe. Vue sous un certain angle, elle est l'un des organismes les plus progressistes du genre dans le monde, pour ce qui touche aux maladies dues à l'amiante. Ce point de vue est valable : la Commission des accidents du travail de l'Ontario a été la première en Amérique du Nord à accorder une indemnité pour le cancer du poumon chez les travailleurs de l'amiante qui ne présentaient pas également les signes cliniques de l'amiantose; la CAT est le chef de file en ce qui concerne l'indemnisation des travailleurs de l'amiante atteints de cancer du larynx et de cancer gastro-intestinal; elle a ouvert la voie dans le domaine de la réadaptation et des mesures d'extension visant à identifier les travailleurs qui auraient pu souffrir de maladies dues à l'exposition professionnelle à l'amiante. Sous l'autre angle, la CAT paraît arbitraire et capricieuse, dépourvue de direction du sommet à la base et sans équité dans sa procédure. Malheureusement, ce dernier point de vue nous semble éminemment valable.

Il y a une explication à ce paradoxe issu d'orientations incompatibles et pourtant valables. De par sa loi constitutive, la Commission des accidents du travail est dotée d'un pouvoir discrétionnaire étendu qu'elle a employé à des fins éclairées. Cependant, ce même pouvoir discrétionnaire est le berceau de l'arbitraire parce que la CAT n'a pas la structure appropriée et l'encadrement nécessaire.<sup>47</sup> Une partie du problème vient de la loi et non de

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<sup>47</sup>Ce thème revient constamment tout au long des chapitres 12 et 13.



l'organisme qui en dépend. Nous endossons donc avec ferveur les recommandations du professeur Weiler qui envisagent la création d'un Tribunal d'appel des décisions concernant les accidents du travail, lequel serait soutenu par des Comités de révision médicale et rendrait par écrit des décisions argumentées. Comme le professeur Weiler, nous conseillons instamment de modifier la loi de sorte que le Conseil de direction de la CAT puisse se composer en majorité de commissaires externes à temps partiel, mais à la différence du professeur Weiler, nous prescrivons d'augmenter un peu plus le nombre de membres.<sup>48</sup>

Bien que le problème de la CAT soit imputable en partie à la loi, l'autre part est de sa propre fabrication. Dans le domaine précis des maladies associées à l'amiante, la CAT a négligé de recourir à un article de sa loi constitutive, lequel crée une présomption légale en faveur des réclamants dont la maladie est directement associée à l'exposition à un procédé industriel.<sup>49</sup> Étant donné que nous considérons le mésothéliome et l'amiantose comme des maladies spécifiques à l'amiante, nous recommandons que le lien entre ces maladies et l'exposition à l'amiante soit garanti par présomption légale et que cette présomption, à présent réfutable, soit rendue irréfutable.<sup>50</sup>

Alors que la Commission des accidents du travail choisissait délibérément, il y a une trentaine d'années, de ne pas user de la présomption légale inscrite dans sa loi constitutive, elle a néanmoins cherché à structurer son pouvoir discrétionnaire en élaborant des directives relatives aux maladies professionnelles. Les réclamants qui satisfaisaient aux conditions stipulées dans les directives obtenaient automatiquement une indemnité, pourvu que les faits exposés dans leur demande fussent véridiques; quant aux demandes qui ne cadraient pas avec les directives, elles étaient examinées minutieusement, cas par cas. Nous avons passé en revue le processus d'établissement des directives, par rapport aux maladies liées à l'amiante, et nous constatons qu'il a été informe, interne, incohérent et incomplet et qu'il n'a contribué en rien à chasser l'impression que la CAT agit de façon arbitraire.<sup>51</sup> Nous recommandons que les règles d'admissibilité soient dorénavant formulées avec le concours d'un nouveau Conseil consultatif pour la politique relative aux maladies professionnelles (CCPRMP), lequel serait autorisé à créer des comités de spécialistes chargés d'aider à élaborer lesdites règles. Sur un plan plus général, nous recommandons que le président du CCPRMP soit nommé un des commissaires au Conseil de direction de la CAT et que le CCPRMP ait un mandat englobant tous les aspects de

<sup>48</sup>Nos vues concernant les appels et la structure du Conseil de direction de la CAT sont élaborées au chapitre 12, section C.1.

<sup>49</sup>Pour plus d'explications, voir chapitre 12, section B.2.

<sup>50</sup>Pour plus d'explications, voir chapitre 12, section C.2.

<sup>51</sup>Nous étayons ces constatations au chapitre 12, section B.3.

la politique relative aux maladies professionnelles. Nous recommandons en outre que ce mandat précise exactement le rôle du CCPRMP quant à la révision des programmes de réadaptation, quant à la surveillance et à l'amélioration des mesures d'extension, et quant à la recherche de méthodes appropriées pour financer les indemnités de maladie professionnelle.<sup>52</sup>

Parmi les nombreuses recommandations touchant les questions d'indemnisation reliées à l'amiante, nous soulignons, aux fins de cet aperçu général, celles qui visent les problèmes de procédure et de fond soulevés par les prestations aux victimes de l'amiantose.<sup>53</sup> Cette maladie irréversible et habituellement progressive, qui peut être fatale en soi et à coup sûr réduit sensiblement l'espérance de vie, est un exemple classique de la maladie professionnelle dont les victimes ont droit à des prestations pour invalidité partielle. Depuis des dizaines d'années, la CAT a recours à un comité de médecins expérimentés en matière de maladies respiratoires, appelé à présent le Comité consultatif des pneumopathies professionnelles (CCPP), pour établir le diagnostic des réclamants et déterminer le quantum (pourcentage) de diminution de capacité de gain chez chaque patient. Le quantum de diminution capacitaire est établi d'après le degré de dégradation physique que la médecine considère cliniquement mesurable. Nous examinons dans le détail le fait que la médecine en est arrivée à reconnaître l'existence d'une diminution psychologique aussi bien que physique. Ensuite, nous passons en revue les évidences, du côté de la littérature médicale et du côté des témoignages faits sous serment, qui démontrent que le diagnostic d'une maladie comme l'amiantose entraîne des répercussions psychologiques néfastes pour le patient. Nous examinons aussi le fait que les tribunaux, dans leur calcul des dommages-intérêts adjugés en responsabilité civile, ont reconnu la dégradation psychologique malgré les difficultés extrêmes que peut causer son évaluation. Nous fondant sur toutes ces observations, nous concluons qu'il faut indemniser les victimes de l'amiantose en fonction de la diminution capacitaire globale, c'est-à-dire tant psychologique que physique. Sur directive émanant du Conseil de direction de la CAT, le CCPP devrait dorénavant classer chaque réclamant, à la lumière d'un diagnostic clinique, par catégorie de diminution capacitaire physique. Trois catégories, ainsi que la littérature médicale internationale le reconnaît, devraient être utilisées pour qualifier la diminution capacitaire : légère, moyenne, grave. Le Conseil de direction de la CAT, et non pas le CCPP, devrait stipuler dans une directive le pourcentage de diminution reconnu aux fins d'indemnisation. Ce pourcentage devrait reconnaître la diminution capacitaire physique et psychologique globale des victimes de l'amiantose conformément à la catégorie de diminution physique dans laquelle le CCPP les aura classées, et il sera de 30% dans les cas

<sup>52</sup>La structure et le rôle du CCPRMP sont exposés au chapitre 12, section C.3; son rôle est élaboré davantage au chapitre 14, voir les parties finales des sections A, B et C.

<sup>53</sup>Tous les points résumés dans ce paragraphe sont élaborés au chapitre 13, section B.

jugés *légers* par le CCPP, de 60% dans les cas jugés *moyens*, et de 100% dans les cas jugés *graves*.<sup>54</sup> Outre cette question de fond, nous nous penchons sur de nombreuses questions de procédure que posent le fonctionnement du CCPP et ses relations avec le Conseil de direction de la CAT, et nous faisons les recommandations que nous jugeons appropriées.

Sur un plan plus général, nous prenons note du fait que la Commission des accidents du travail a le mérite de se guider sur un principe du bénéfice du doute pour prendre ses décisions, de faire des observations quant à savoir dans quelle mesure ce principe n'a été ni appliqué uniformément ni communiqué uniformément, et de conclure que le principe du bénéfice du doute devrait être porté au niveau de la *Loi sur les accidents du travail*.<sup>55</sup> Nous nous attaquons également aux problèmes qui gênent la qualité des communications de la CAT avec les réclamants et faisons des recommandations en conséquence.<sup>56</sup>

En 1975, la Commission des accidents du travail a lancé un programme innovateur pour réadapter les employés dont l'exposition antérieure à l'amiante semblait justifier de les soustraire à une plus ample exposition.<sup>57</sup> Nous évaluons les facteurs qui expliquent pourquoi ce programme a été marqué par la controverse et nous constatons que ses lacunes étaient véritables, mais par ailleurs compréhensibles. Cependant, nous déplorons de constater que les dispositions que l'on retrouve dans le règlement concernant l'amiante à l'égard du retrait et de la réadaptation invitent à répéter les mêmes erreurs, ou à faire pis, et nous formulons des recommandations fondées sur les leçons que l'on a pu tirer du Programme d'aide spéciale à la réadaptation créé par la CAT.

L'effort d'extension est du domaine des mesures visant à identifier les personnes dont la maladie ou la mort est causée par une exposition professionnelle et fait donc l'objet d'une indemnisation en vertu de la loi.<sup>58</sup> Nous envisageons le fait que des décès reliés à l'exposition professionnelle à l'amiante peuvent se produire en Ontario sans faire l'objet d'une indemnisation, et à la lumière de ce fait, nous jugeons nécessaires et importantes les mesures d'extension. Nous examinons ensuite les mesures d'extension prises par la CAT et, bien que nous les trouvions louables, nous concluons que les mesures de ce genre devraient à l'avenir incomber conjointement à la CAT et au ministère du Travail.

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<sup>54</sup>Cette recommandation a des répercussions sur l'admissibilité à des indemnités de décès pour les survivants des malades qui souffraient de l'amiante avant leur mort; la question des règles d'admissibilité en cette matière est examinée au chapitre 13, section C.

<sup>55</sup>Ces points sont élaborés au chapitre 13, section D.

<sup>56</sup>Voir chapitre 13, section E.

<sup>57</sup>Les points résumés dans ce paragraphe sont élaborés au chapitre 14, section A.

<sup>58</sup>Les points résumés dans ce paragraphe sont élaborés au chapitre 14, section B.



Enfin, nous étudions dans quelle mesure un régime d'indemnisation des accidents du travail peut jouer un rôle de prévention à l'égard des maladies professionnelles.<sup>59</sup> Nous constatons que la prévention est étroitement liée à d'autres buts dominants, notamment l'équité chez les employeurs et la justice. Nous examinons les dispositions de la *Loi sur les accidents du travail* et des règlements afférents qui, avec les procédés en pratique à la CAT, régissent le financement des coûts d'indemnisation et l'évaluation des pénalités. Nous constatons que l'expérience due à l'amiante indique des lacunes graves, que ce soit au plan de la prévention, de l'équité ou de la justice, et nous faisons des recommandations pertinentes.

## **G. L'amiante : une expérience riche en enseignements**

Ce rapport tout entier est un exercice d'apprentissage dicté par l'expérience de l'amiante; il faut savoir tirer les leçons qu'il propose à l'égard de la salubrité et de la réglementation. Le rapport se termine par des observations sur ce que l'expérience de l'amiante nous apprend au sujet de l'identification des dangers et du dispositif en place à l'heure actuelle pour désigner les substances dangereuses employées dans les lieux de travail en Ontario.

L'histoire de l'amiante décrit comment le dispositif d'identification des dangers peut se détraquer lentement et comment les fonctions de réglementation peuvent se laisser distancer par l'accumulation progressive des connaissances, alors que la maladie et la mort fauchent un nombre croissant de travailleurs relégués au rang de cobayes.<sup>60</sup> Cet état de choses inacceptable exige une alternative, c'est-à-dire une identification beaucoup plus prompte par de nouvelles techniques de pré-sélection et une réponse de surveillance réglementaire plus prompte. Heureusement, la société reconnaît à présent que des facteurs nés du milieu humain contribuent sensiblement à une mort prématurée, que le traitement médical a ses limites en ce qui concerne les possibilités de guérison dans beaucoup de ces cas, et que les mesures préventives constituent la meilleure approche pour enrayer beaucoup de maladies. Toute récente qu'elle soit, la reconnaissance de ces facteurs par la société a commencé à fructifier et on assiste à l'avènement de méthodes plus efficaces pour identifier les dangers et d'une réglementation plus opportune et plus efficace. Si l'expérience de l'amiante en milieu de travail offre un témoignage sévère quant aux défauts des vues sociales antérieures, marquées par l'indifférence envers la prévention de la maladie, cette expérience proclame également l'importance du discernement dont il

<sup>59</sup>Les points résumés dans ce paragraphe sont élaborés au chapitre 14, section C.

<sup>60</sup>Les points résumés dans ce paragraphe sont élaborés au chapitre 15, sections B et C.



faut faire preuve pour définir les dangers et formuler les solutions réglementaires. Avec les années 1970, la société s'est sensibilisée à l'influence de l'environnement sur la maladie et à l'importance de la prévention. Toutefois, tandis que le public des années 1970 découvrait l'énormité de la catastrophe qui ravageait la santé des travailleurs de l'amiante, on imposait des règlements et on prenait des mesures de protection sans s'apercevoir qu'une substance mortelle en milieu de travail peut poser des risques insignifiants dans un milieu ordinaire à l'intérieur ou à l'extérieur. Cela témoigne en faveur d'une meilleure capacité d'identification des dangers et d'évaluation des risques de la part des pouvoirs publics et nous incite à recommander instamment que soit créé, de préférence à l'échelle nationale, un organisme chargé d'identifier les dangers et d'évaluer les risques, lequel serait doté de moyens scientifiques perfectionnés et aurait toute liberté de communiquer avec les représentants élus, les médias d'information et les corps publics investis d'un pouvoir de réglementation. Nous envisageons aussi la nécessité de mieux coordonner la communication de l'information émanant des organismes chargés de réglementer les situations dangereuses, et nous faisons des recommandations en conséquence.

Une commission royale d'enquête est sans doute le plus malvenu des organes publics à faire des observations concernant la quantité de temps qu'exige toute opération d'ordre public. Nous le faisons néanmoins à l'égard du processus par lequel le ministère du Travail désigne les substances dangereuses à des fins de réglementation.<sup>61</sup> Dans le cas de l'amiante, il a fallu plus de deux ans pour y arriver. La participation ouverte qui caractérise ce processus et explique en grande partie sa marche mesurée est tout à fait louable à notre avis. Pourtant, si l'on veut assurer aux travailleurs davantage de protection pendant ce long processus de désignation, nous croyons que le ministère du Travail doit envisager l'opportunité d'une démarche en deux étapes suivant laquelle on adopterait un règlement préliminaire lors de la publication de l'avis d'intention de désigner et on le garderait en vigueur jusqu'à la promulgation d'un règlement définitif.

Cette Commission royale d'enquête était-elle vraiment nécessaire? Nous revenons sur la question posée au début du chapitre parce qu'il appartient aux lecteurs de ce rapport d'y répondre. Une dernière observation s'impose : la présente Commission royale d'enquête a duré presque quatre ans et elle a coûté 1,7 million de dollars aux contribuables de l'Ontario.

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<sup>61</sup> Les points résumés dans ce paragraphe sont élaborés au chapitre 15, section D.

# **Text of Formal Recommendations**

There follows, numbered sequentially by chapter, the text of the 117 formal recommendations made by this Commission.

## **From: Part III Asbestos in Fixed Workplaces**

### **Chapter 7 Control Limits for Fixed Place Industry**

- 7.1** Whenever control limits that cannot be enforced by present measurement techniques are adopted, the Ministry of Labour should emphasize that section 17 of the Regulation Respecting Asbestos allows the substitution of alternative measurement techniques if the Ministry is convinced that the substitute techniques satisfy the criteria of section 17 of the Regulation.
- 7.2** The Ministry of Labour mounting method should be amended to replace the dimethyl phthalate and dimethyl oxalate preparation step with the acetone-Triacetin preparation step.

- 7.3 The Ministry of Labour should require that all slides and remaining filter segments that represent workplace exposures in Ontario, whether counted by the Ministry of Labour or by a private firm, be catalogued and stored in Ontario for a period of five years after the sample is taken.
- 7.4 The Ministry of Labour method should be modified to incorporate tests of microscope resolution.
- 7.5 The Ministry of Labour should specify the Walton-Beckett graticule exclusively in the Code for Measuring Airborne Asbestos Fibres.
- 7.6 The Ministry of Labour method should be modified to provide a more rigid system of quality assurance, including the use of blank filters and recounting by other operators.
- 7.7 The fibre counting criteria published in the Asbestos International Association's (AIA) reference method should be adopted by the Ontario Ministry of Labour as an interim measure since they represent a current level of international consensus. If and when agreement is reached on counting criteria to be adopted for the International Organization for Standardization/Draft International Standard (ISO/DIS) method, these should then be considered for adoption by Ontario.
- 7.8 The Ministry of Labour should identify a laboratory to take responsibility for standardizing methods of asbestos fibre counting for workplace samples in Ontario and to certify individuals whose performance is judged by this laboratory to be satisfactory. Every attempt should be made to include worker representatives from Ontario asbestos manufacturing plants among such certified operators where the volume of measurement warrants it.
- 7.9 The Ministry of Labour should undertake a study of the relationship between the number of air samples taken and the distribution of those air samples among workers, work stations, and periods of the day or week, in order to determine the minimum requirements for an air sampling programme that will provide reasonable confidence that the data collected adequately reflect the maximum worker exposures in a workplace, the average exposure of those workers, and those work stations where particularly intensive monitoring may be desirable. This study should incorporate such statistical analyses as are necessary for the task. Upon completing the study, the Ministry should amend the Regulation Respecting Asbestos to provide more specific direction regarding the sampling programme that will satisfy that Regulation.

- 7.10 The Ministry of Labour should conduct research using a scanning electron microscope alongside an optical microscope to explore the size distribution of fibres found in various workplaces.
- 7.11 The Government of Ontario should adopt regulations that would prohibit the operation of asbestos textile spinning and weaving plants in Ontario without the approval of the Ministry of Labour. This approval should be granted only if the Ministry is satisfied that the health risks faced by workers in the plant are far lower than the unacceptable risks estimated in this Report.
- 7.12 The Government of Ontario should adopt regulations that would prohibit the use of crocidolite asbestos in Ontario workplaces without the approval of the Ministry of Labour. This approval should be granted only if the Ministry is satisfied that the health risks faced by workers exposed to this asbestos are far lower than the unacceptable risks estimated in this Report.
- 7.13 The Code for Respiratory Equipment for Asbestos on Construction Projects should provide for the use of respiratory protection that will ensure that the worker's exposure does not exceed 0.02 fibres per cubic centimetre where crocidolite asbestos is in the air.
- 7.14 The Government of Ontario should adopt a regulation which prohibits the use of amosite in Ontario manufacturing unless and until it is demonstrated to the satisfaction of the Ministry of Labour that there is a measurement technology that will satisfactorily detect low amosite concentrations so that a 0.1 fibre control limit can be enforced with a reasonable degree of confidence.
- 7.15 The Code for Respiratory Equipment for Asbestos on Construction Projects should provide for the use of respiratory protection that will ensure that the worker's exposure does not exceed 0.1 fibres per cubic centimetre where amosite asbestos is in the air.
- 7.16 The Ministry of Labour, in applying the control limits of the Regulation Respecting Asbestos to automotive brake repair operations, should accept the use of wet methods of brake drum cleaning as *prima facie* evidence that the Regulation has been complied with.
- 7.17 The Ministry of Labour, in applying the control limits of the Regulation Respecting Asbestos to exposures of workers engaged in grinding and bevelling of brake drums in automotive brake repair operations, should accept as evidence that the Regulation is complied with:



- (i) that the operator is wearing a respirator while grinding and while cleaning the debris afterwards with wet methods, in shops where this operation occurs rarely; and
  - (ii) that the machines are equipped with local exhaust ventilation, in shops where grinding of brake shoes occurs frequently.
- 7.18 The Regulation Respecting Asbestos should be amended to specify that sections 6 through 16 do not apply to workplaces where the only exposure to asbestos arises out of the repair and maintenance of automotive brakes, unless grinding of brake shoes is performed frequently.
- 7.19 The Ministry of Labour should develop a modest information programme to communicate with the vast number of auto mechanics in Ontario the appropriate work practices as described in Recommendations 7.16, 7.17, and 7.18. A more intensive programme of communication should be designed to reach those mechanics working in brake specialty shops where, in particular, a considerable volume of grinding of new brake shoes is performed. The Ministry should also communicate appropriate practices to the secondary and post-secondary institutions in Ontario where auto mechanics training is currently provided.

## **Chapter 8 Beyond Control Limits: Protecting Health in Fixed Place Industry**

- 8.1 The Ministry of Labour should undertake to identify instances of the successful operation of joint health and safety committees and the means whereby the factors that account for this success can be disseminated through information and training programmes, drawing upon the review by the Advisory Council on Occupational Health and Occupational Safety and such other analyses as are necessary.
- 8.2 The *Workers' Compensation Act* should be amended to provide that where an employer establishes or contributes from his own resources to the financing of a joint labour-management programme for the purpose of education in accident or disease prevention, and the Board is satisfied that the programme is serving the same purpose as an Accident Prevention Association programme, the Board may reduce the assessment for which the employer is liable as a member of a class represented by an Association.

- 8.3 The *Occupational Health and Safety Act* should be amended to provide that, where stipulated by regulation, members of a joint health and safety committee may be entitled to paid time from work for the purpose of occupational health and safety training; the Regulation Respecting Asbestos should be amended to stipulate that members of joint health and safety committees shall be entitled to paid time from work for the purpose of occupational health training in programmes approved by the Minister.
- 8.4 The Regulation Respecting Asbestos should be amended so as to require that every asbestos worker shall be informed of:
- (i) the existence of a dose-response relationship for asbestos-related disease, namely, the lower the exposure, the lower the risk of disease; and
  - (ii) the fact that the Regulation establishes control limits as maxima and imposes on the employer the duty to take all necessary measures and procedures to ensure that the time-weighted average exposure of a worker to airborne asbestos is reduced to the lowest practical level.
- 8.5 Section 12 of the Regulation Respecting Asbestos should be amended to require that each time monitoring of airborne asbestos takes place, an appropriate summary statistic of the resulting fibre concentrations be calculated for each area of the plant, where areas are defined as portions of the plant in which dust levels are reasonably similar. The results for each area to be posted under section 12 should include these summary statistics. In addition, a summary of the average dust level in each area over the past one or two years should be calculated and compared with the current dust levels and with a weighted average of recent dust levels. The results of the monitoring and of this analysis should be posted as specified in section 12(a), but for a period of at least two months, or until rendered obsolete by subsequent measurements, rather than for the fourteen-day period specified in the existing Regulation.
- 8.6 The Regulation Respecting Asbestos should be amended so as to require that in every plant with a control programme, there shall be an individual certified by the Occupational Health Hygiene Service who will be designated as responsible for designing representative air sampling procedures; for seeing that the Code for Measuring Airborne Asbestos Fibres is respected; and for ensuring that equipment is properly calibrated.

**8.7 The Regulation Respecting Asbestos should be amended to require that the worker representative appointed pursuant to section 8(8) of the *Occupational Health and Safety Act*:**

- (i) have the opportunity to observe, on his plant tour, the calibration of air monitoring equipment;
- (ii) be consulted on the adequacy of representative air sampling procedures; and
- (iii) be afforded preparation time, together with office and secretarial assistance, beyond that already recognized for the preparation of minutes of committee meetings.

**8.8 The Regulation Respecting Asbestos should be amended so as to require that every employer with a control programme must select persons who have supervisory positions for training in the nature of the hazards posed by asbestos, and in identifying and dealing with the specific workplace situations that may be most hazardous. The number of persons trained should be sufficient to ensure that one such person will be on duty in the plant at all times when its operation or maintenance entails worker exposure to asbestos.**

**8.9 The Ministry of Labour should review the content of the basic occupational health training it offers to Industrial Health and Safety Branch inspectors with a view to its adequacy and to whether it might be integrated with available joint labour-management health training programmes, and the Ministry should seek as expeditiously as possible to enhance the proportion of its inspectorate that has received occupational health training. As an interim measure, the Ministry should make every effort to ensure that the inspectors assigned to plants where designated substances are present are individuals who have received basic occupational health training.**

**8.10 The Ministry of Labour should create a Designated Substances Enforcement Unit (DSEU), headed by an official with the rank of Branch Director. This official would have the authority to launch special, unannounced inspection visits of plants in which designated substances are in use and to commandeer a multidisciplinary team of appropriate Ministry experts assembled separately for each inspection visit. Each DSEU team should have all the powers of a Ministry of Labour inspector.**

**8.11 The Regulation Respecting Asbestos should be amended to clarify that the examining physician under sections 13 to 16 shall be the physician designated by the employer, but that any employee shall**

have the right to be examined, at the expense of the employer, by a physician selected by the employee, provided this physician certifies to the employer that he has conducted the examination in accordance with the Code for Medical Surveillance of Asbestos Exposed Workers.

- 8.12 The Regulation Respecting Asbestos should be amended so as to substitute for the role of examining physicians in maintaining the records, referred to in section 15(1), an obligation on examining physicians to forward the records or copies thereof to the Chief Physician, Occupational Health Medical Service of the Ministry of Labour. The system of control pursuant to which the Ministry maintains these records should be that prescribed by the Ontario Commission of Inquiry into the Confidentiality of Health Information.

## **From: Part IV Asbestos in Buildings**

### **Chapter 9 Problems of Asbestos in Buildings**

- 9.1 The Ministry of Labour should prohibit the spraying of friable material containing more than 1% asbestos on a dry-weight basis.
- 9.2 The Ministry of Labour should assess, sponsoring studies where appropriate, the health risks associated with the fibres used as substitutes for asbestos in sprayed insulation, with a view to determining what regulations, *if any*, might be appropriate for the use of these substitutes.
- 9.3 The Ministry of Labour should ban the application of friable materials containing more than 1% asbestos by weight as insulation for pipes and boilers.
- 9.4 The Ministry of Labour should identify the compounds used as substitutes for pipe and boiler insulation, with a view to assessing any health risks associated with these substitutes and to determining what regulations, *if any*, might be appropriate for the use of these substitutes.
- 9.5 Legislation should be developed providing that, if significant quantities of friable insulation have fallen onto building surfaces and are being disturbed, the building owner must have the material tested to



determine whether it contains asbestos. This requirement should apply only to insulation installed or replaced before 1974.

- 9.6 Legislation should be developed providing that if the test mandated by the circumstances stipulated in Recommendation 9.5 shows that friable asbestos is present, the building owner must enclose, encapsulate, or remove the asbestos-containing material, following the work practices referred to in Recommendation 10.17.
- 9.7 The Minister of Labour should communicate with the Minister of Health with a view to establishing responsibility with respect to Recommendations 9.5 and 9.6.
- 9.8 Owners of private homes should be exempt from the duties set out in Recommendations 9.5 and 9.6 above.
- 9.9 The Ministry of Labour should communicate with workers who replace and maintain domestic space-heating boilers to inform them of the risks of working with asbestos and of the appropriate safety precautions for such work.
- 9.10 The Ministry of Labour should prepare a brief flyer describing the possible locations of asbestos on pipes and boilers in the home, the proper method of sampling suspect material, and any appropriate precautions should asbestos be found to be present.

## Chapter 10 Protecting Workers from Asbestos in Buildings

- 10.1 The Ministry of Labour should frame a regulation under the *Occupational Health and Safety Act* respecting asbestos on construction projects and in buildings which includes building custodial and maintenance work. The Ministry should amend the existing Regulation Respecting Asbestos so as to specify that its terms do not apply to work activities encompassed by the new regulation.
- 10.2 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require as follows:
- (i) Before the demolition or renovation of any building the characteristics of which raise the possibility that it might contain friable asbestos, the building owner must cause the building or relevant part of the building to be inspected to determine

whether friable asbestos-containing materials are present. If friable asbestos-containing materials are present, Recommendation 10.11 should be followed in the case of demolition, and Recommendations 10.14 and 10.17 should be followed in the case of renovation.

- (ii) Before performing maintenance or custodial work which would disturb friable fibrous material in any building the characteristics of which raise the possibility that it might contain asbestos, the building owner must cause the material to be disturbed to be tested for asbestos content. If the material is found to contain asbestos, the employer must cause work practices to be adopted which ensure the health and safety of the maintenance or custodial workers, as outlined in Recommendations 10.6, 10.14, and 10.17. Alternatively, the maintenance or custodial work may proceed without the suspect material being tested, provided that the work practices followed would be safe in the event that the material did contain asbestos.
- (iii) The requirements of (i) and (ii) above should apply only to buildings in which insulation was installed or replaced before 1974.

**10.3** The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require that in any case where a building is required by law to be inspected or tested for asbestos, the building owner must cause the results of the inspection or testing to be recorded as follows:

- (i) in a public place to be designated by the Ministry of Labour;
- (ii) in a document maintained on the building premises, under the care of the building owner or his representative. This document should be required to be shown on request to any government inspector, any building occupant, or any worker who is in the building for the purpose of exercising his trade and who might be exposed to friable fibrous materials; and
- (iii) by placing signs or labels, in areas where asbestos material has been discovered, that clearly indicate the presence of asbestos and warn that the material should not be disturbed without using appropriate precautions.

**10.4** The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should provide for standardized laboratory procedures to be followed for analysis of bulk material taken from buildings. These procedures should be

reviewed from time to time so as to keep up with developing technology.

- 10.5 The Ministry of Labour should take steps to institute a register of individuals certified as competent to inspect buildings for asbestos. Certificates of competence should be awarded by the Ministry of Labour upon an applicant's having satisfactorily completed an approved examination. Incompetence or dishonest practices should result in the certificate being revoked. Only individuals named in the register should be permitted to undertake inspections for asbestos in buildings which are required by law.
- 10.6 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require that once friable asbestos-containing material is found in a building, the building owner must institute a programme of management and custodial control. The scope of the programme should depend on the level of hazard created by the asbestos-containing material and should provide for the institution of safe work practices, including worker training therein, for all regular building custodial and maintenance workers. The programme should continue until the asbestos-containing material is removed from the building.
- 10.7 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require that a written description of any management and custodial control programme, referred to in Recommendation 10.6 above, be submitted to the Ministry of Labour for approval. The Ministry of Labour should be empowered to approve or vary any programme so submitted, in order to ensure adequate protection for building workers.
- 10.8 The regulation pursuant to the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should govern the operations of a contractor who contracts with a building owner to take responsibility for worker training and safe work practices under a management and custodial control programme, so that the contractor would have the same responsibility as the building owner for ensuring that the relevant terms of the management and custodial control programme are met.
- 10.9 The Ministry of Labour should take steps to require that every contract for renovation, maintenance, or custodial work specify all known locations of friable asbestos-containing materials in the building which might be disturbed. If, in the course of the work, friable asbestos-containing materials of which the contractor was not informed are discovered in locations which could create some risk to

workers, adequate precautions for worker protection should be taken, at the sole expense of the building owner.

- 10.10 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should provide that where removal, enclosure, or encapsulation of sprayed asbestos-containing insulation is contemplated, encapsulation should only be an option if that insulation is in good condition and is sufficiently thin that it can be thoroughly penetrated by the encapsulant, or if removal or enclosure are not practicable.
- 10.11 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require that the owner of a building or structure ensure that prior to the demolition or partial demolition of a building or structure, all friable asbestos-containing materials which might be disturbed are removed from the building or structure, following procedures for removal referred to in Recommendations 10.14 and 10.17.
- 10.12 The Ministry of Labour should take steps to require that every contract for demolition specify all known locations of friable asbestos-containing materials in the building or structure which might be disturbed by the demolition work.
- 10.13 The Ministry of Labour should take steps to provide that all contracts for the demolition of a building or structure in Ontario shall be deemed to include a clause which provides for supplemental payment to the demolition contractor, on a time and materials basis, for all reasonable expenses incurred in removing any friable asbestos-containing material which was not specified in the demolition contract.
- 10.14 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should distinguish among three levels of precautions:

**“Precautions A”:**

The minimum precautions, or “Precautions A,” should include the following:

- (i) Workers must wear respirators in the hazardous work area [section 6, Proposed Regulation Respecting Asbestos on Construction Projects (PRRACP)].
- (ii) Power tools used in the hazardous work area on asbestos-containing material may be operated only in conjunction with



high efficiency particulate air filtered vacuums (section 9, PRRACP).

- (iii) Workers must be educated as to the risks they face when working with asbestos and trained in the appropriate procedures for minimizing those risks (similar to section 11, PRRACP).

**“Precautions B”:**

The intermediate precautions, or “Precautions B,” should include, in addition to the precautions set out above as “Precautions A,” the following:

- (i) The Ministry of Labour must be notified before the hazardous work begins (section 4, PRRACP).
- (ii) The work area must be separated from the rest of the project by a barrier capable of limiting the spread of asbestos fibres outside the work area (to be added to PRRACP).
- (iii) Protective clothing must be worn by all workers (section 7, PRRACP).
- (iv) Washing facilities must be available to workers (section 7, PRRACP).
- (v) The hazardous work area must be properly and regularly cleaned (section 8, PRRACP).
- (vi) No loose asbestos is to remain on the floors (section 10, PRRACP).
- (vii) Asbestos-containing materials must be wetted prior to disturbing them, where practicable (section 13, PRRACP).
- (viii) Asbestos work records must be kept for all workers involved in the hazardous work (section 14, PRRACP).
- (ix) All workers involved in the hazardous work are to be subject to medical surveillance (sections 15–18, PRRACP).

**“Precautions C”:**

The maximum precautions, or “Precautions C,” should include, in addition to the precautions set out above as “Precautions ‘A’ and ‘B,’ ” the following:

- (i) The work area must be enclosed and sealed (section 13, PRRACP).

- (ii) Showers and change rooms must be available to all workers (section 13, PRRACP).
  - (iii) The work area must be cleaned prior to the commencement of the work and subjected to a thorough two-stage cleanup after the work is completed (section 13, PRRACP).
- 10.15 The Ministry of Labour should consult with industry and labour to develop numerical definitions of areas of insulation to be classified as “small,” “medium,” and “large” for the purpose of determining which level of precautions, whether “Precautions ‘A,’ ‘B,’ or ‘C’ ” referred to in Recommendation 10.14 above, is to be applied to asbestos control work projects of varying types and sizes.
- 10.16 The Ministry of Labour should consult with industry and labour to develop definitions of major, intermediate, and minor renovation, maintenance, and custodial projects for the purpose of determining which level of precautions, whether “Precautions ‘A,’ ‘B,’ or ‘C’ ” referred to in Recommendation 10.14 above, is to be applied in each instance.
- 10.17 The Ministry of Labour should consult with industry and labour to determine, for the purposes of the regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings, the appropriate precautions to be applied to large, medium, and small control jobs and to major, intermediate, and minor renovations and maintenance and custodial projects, distinguishing, where appropriate, among wet-sprayed insulation, dry-sprayed insulation, and pipe and boiler insulation.
- 10.18 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require that types of work in a hazardous work area which are not otherwise dealt with in that regulation shall only be performed in accordance with “Precautions A,” described in Recommendation 10.14.
- 10.19 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should provide that where a worker frequently performs work calling for “Precautions A,” described in Recommendation 10.14, asbestos work records should be kept for that worker and that worker should be subject to medical surveillance, as if the work called for “Precautions B.”
- 10.20 The Ministry of Labour should, in consultation with industry and labour, prepare an alternative set of precautions for asbestos removal work outdoors that would allow the substitution of local

exhaust ventilation for the use of an enclosure in situations where enclosure is impracticable. This alternative set of precautions should be included in the regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings.

- 10.21 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should provide that work procedures may be used which vary from those prescribed if the protection afforded by the variant procedures is equal to or exceeds the protection afforded by the prescribed procedures, or if it is demonstrated to the Ministry of Labour that those variant work procedures would always result in average worker exposures which, in the case of chrysotile, are at or lower than one-half the control limit and, in the case of crocidolite or amosite, are at or lower than one-half the exposure values prescribed in Recommendations 7.13 and 7.15. However, the use of variant work procedures should not in any way detract from requirements that would otherwise apply to the particular work situations regarding worker training, Ministry of Labour notification, asbestos waste handling, cleaning of the hazardous work area, wetting of asbestos, asbestos work records, or medical surveillance.
- 10.22 The Ministry of Labour should conduct air monitoring of control work, renovation work, and of maintenance and custodial work, from time to time, to determine whether the safety precautions referred to in Recommendations 10.14, 10.17, and 10.20 above are sufficient to ensure worker safety or are unnecessarily strict.
- 10.23 The Ministry of Labour should explore methods of communicating with control, renovation, maintenance, and custodial workers to inform them of the risks they face when working in buildings containing friable asbestos and of the appropriate procedures for minimizing those risks.
- 10.24 The Ministry of Labour should take steps to institute a register of individuals certified as competent to supervise the enclosure, encapsulation, or removal of asbestos-containing sprayed insulation and pipe and boiler insulation. Certificates of competence should be awarded by the Ministry of Labour; incompetence should result in the certificate being revoked.
- 10.25 The Ministry of Labour should take steps to require that no asbestos enclosure, encapsulation, or removal may take place without being supervised by a registered work supervisor. The Ministry of Labour should determine what exceptions to this requirement should be made for small jobs.

## **From: Part V Asbestos Elsewhere**

### **Chapter 11 Asbestos in the Environment**

- 11.1 The Government of Ontario should take steps, either through federal-provincial collaboration or by having recourse to provincial legislative jurisdiction, towards the regulation of asbestos-containing consumer and construction products in accordance with the following scheme:**

**Products containing asbestos should be classified into one of three categories:**

**Category One:**

**This category would embrace loose asbestos which, during normal use or handling, is likely to release asbestos fibres in significant concentrations. An example of this is loose-fill asbestos insulation.**

**Category Two:**

**This category would embrace products containing asbestos which possess the potential to release asbestos fibres in significant concentrations in the event of misuse, improper handling, manipulation such as cutting or sanding, or as a result of product degradation. Products in this category would include asbestos-cement sheet; and asbestos gloves, simmering pads, and ironing board replacement sheets.**

**Category Three:**

**This category would embrace products containing asbestos which is sealed off or encapsulated so that the release of asbestos fibres is unlikely under foreseeable use. Products in this category would include most appliances which contain asbestos insulation and molded plastic products containing asbestos filler.**

- 11.2 The following regulatory actions should be taken with respect to Categories One and Two:**

- (i) The manufacture, sale, and use of products in Category One, other than essential products for which there is no adequate substitute, should be prohibited in Ontario, unless the product is exclusively for the use of a manufacturing enterprise governed by the existing Regulation Respecting Asbestos.**



- (ii) All products in Category One, the manufacture, sale, or use of which are not prohibited in Ontario, and all products in Category Two, should be labelled with the following warning:  

**“Caution — Contains Asbestos — Breathing Asbestos May Be Dangerous to Your Health — Consult Pamphlet Available from Vendor for Proper Use.”**
  - (iii) All purchasers of construction materials labelled in accordance with (ii) above should be provided with a pamphlet outlining the nature of the hazard posed by the product and the appropriate precautions to be taken in connection with the product.
- 11.3 Products in Category Three should not be subject to regulation.
  - 11.4 The Ministry of Consumer and Commercial Relations should take steps to repeal the Liquor Control Board of Ontario ban on the use of asbestos filters.
  - 11.5 The Ministry of the Environment, in co-operation with the Ministry of Labour, should financially support the development of internationally accepted methods for ambient air quality measurement and adopt such measurement methods when they have been adopted by such international bodies as the International Organization for Standardization.
  - 11.6 The Ministry of the Environment should revise the emission standard for asbestos to specify concentrations of fibres longer than 5 microns. This standard should be consistent with the existing ambient air quality criterion for asbestos.
  - 11.7 The Ministry of the Environment should amend the 1983 General — Waste Management Regulation made under the *Environmental Protection Act* so as to provide that the sections regulating asbestos waste apply only to asbestos waste generated by asbestos removal operations and by asbestos product manufacturing.
  - 11.8 The Ministry of the Environment should amend the 1983 General — Waste Management Regulation made under the *Environmental Protection Act* so as to require that the Ministry be notified, prior to the transporting of asbestos waste, of the quantity of the waste, the nature and destination of the waste, the type of packing involved, and any other pertinent details.
  - 11.9 The Ministry of the Environment should amend the 1983 General — Waste Management Regulation made under the *Environmental Protection Act* so as to require that precise records be kept as to the

location of deposited asbestos waste. The content and storage of these records should be specified by the Ministry.

- 11.10 The Ministry of the Environment should determine which disposal site operators are prepared to accept asbestos waste. If this determination indicates that there are not adequate disposal sites for receiving asbestos waste in the province, the Ministry of the Environment should either establish special asbestos disposal sites or consider requiring certain disposal site operators to accept asbestos waste.
- 11.11 The Ministry of the Environment should develop policies regarding the redevelopment of waste sites known to contain substantial quantities of asbestos.
- 11.12 The Ministry of the Environment should, where reasonably practicable, endeavour to identify sites in which substantial quantities of asbestos have been deposited.

## **From: Part VI Compensating Victims: Asbestos and Its Implications**

### **Chapter 12 Determining Eligibility for Asbestos-Related Disease Compensation**

- 12.1 In line with the legislative exposure draft in the *White Paper on the Workers' Compensation Act*, the Corporate Board of the Workers' Compensation Board should be composed of a majority of outside directors.
- 12.2 Contrary to the legislative exposure draft in the *White Paper on the Workers' Compensation Act*, the number of Corporate Board members should be fixed at a maximum of twelve.
- 12.3 The provisions of section 80 of the legislative exposure draft in the *White Paper on the Workers' Compensation Act* concerning access to medical reports and opinions should be enacted in substantially their present form.

- 12.4 The provisions of the legislative exposure draft in the *White Paper on the Workers' Compensation Act* concerning the structure and procedures of the "Workers' Compensation Appeals Tribunal" should be enacted in substantially their present form.
- 12.5 The provisions of section 75(3) of the legislative exposure draft in the *White Paper on the Workers' Compensation Act* concerning the requirement that Appeals Tribunal decisions, including findings and reasons, be made in writing should be enacted into law, together with the supporting provisions requiring communication of decisions to the claimant, employer, or party of record.
- 12.6 The provisions of the legislative exposure draft in the *White Paper on the Workers' Compensation Act* concerning the structure and procedures of Medical Review Panels should be enacted in substantially their present form.
- 12.7 The Workers' Compensation Board, at the earliest opportunity, should advise the Lieutenant Governor in Council to amend Schedule 3 of the *Workers' Compensation Act* by inserting in the first column (the disease column) the words: "Asbestosis and Mesothelioma"; and in the second column (the process column) the words: "Any process involving the use of asbestos."
- 12.8 The Workers' Compensation Board should review Schedule 3 of the *Workers' Compensation Act* to ensure that it incorporates those diseases whose associated industrial process is necessary and sufficient to cause the disease.
- 12.9 In line with the legislative exposure draft in the *White Paper on the Workers' Compensation Act*, the provisions of Schedule 3 should be applicable to workers who have engaged in a scheduled industrial process at any time in their employment history.
- 12.10 Section 122(9) of the *Workers' Compensation Act* should be amended so as to stipulate an irrebuttable presumption in favour of the claimant.
- 12.11 The *Workers' Compensation Act* should be amended to provide for the creation of an Advisory Council on Industrial Disease Policy responsible for advising the Corporate Board on eligibility rules regarding disease compensation and on other matters of industrial disease policy. The Council's advice should be developed either as a matter of its own initiative or in response to an explicit request from the Corporate Board. The Council should be appointed by the Lieutenant Governor in Council and be composed of a Chairman, who would be an *ex officio* Director of the Corporate Board, and

not more than eight members appointed for stated terms. The legislation should authorize the Council to appoint expert Panels from time to time and should stipulate that both the Council's advice and the Corporate Board's response be public documents.

12.12 The Advisory Council on Industrial Disease Policy should, as a matter of high priority, transform the current Board guidelines on asbestos-related cancers into eligibility rules in accordance with the process prescribed in this Report.

12.13 The *Workers' Compensation Act* should be amended so as to:

- (i) entitle individuals who contract mesothelioma, and are family members of asbestos workers who were domiciled with these workers at the time such workers were occupationally exposed to asbestos, to the same compensation benefits as the Act accords to employees; and
- (ii) vest in the Workers' Compensation Board a statutory right to recover the costs of benefits paid to a family member of an asbestos worker from the employer of that worker.

## Chapter 13 Processing Asbestos-Related Claims: Procedural and Substantive Issues

13.1 The Workers' Compensation Board, by directive of the Corporate Board, should promulgate as a matter of Board policy a rule for determining the quantum of compensable physical and psychological impairment arising from asbestosis in claimants diagnosed by the Advisory Committee on Occupational Chest Diseases (AC OCD) as suffering from this disease. The rule should instruct the AC OCD to assign such claimants to one of three classes of physical impairment defined by competent medical authorities as corresponding to Mild, Moderate, and Severe respiratory impairment and direct that the percentages to be used by the Claims Adjudication Branch for calculating awards for physical and psychological impairment be:

- (i) where the AC OCD finds Mild physical impairment, 30%;
- (ii) where the AC OCD finds Moderate physical impairment, 60%; and
- (iii) where the AC OCD finds Severe physical impairment, 100%.



- 13.2 In assessing the implications of Recommendation 13.1 for the compensation of other diseases, the Workers' Compensation Board should address those diseases whose associated industrial process is their necessary and sufficient cause and whose manifestation involves chronic, irreversible disorders that shorten the individual's life expectancy.
- 13.3 The statutory definition of impairment contained in the legislative exposure draft in the *White Paper on the Workers' Compensation Act* should be reviewed to ensure that the statutory language of any relevant amendment to the *Workers' Compensation Act* is couched in language that includes psychological impairment.
- 13.4 The Corporate Board should promulgate a rule applicable to the procedural adjudication of asbestosis claims which stipulates that a physician of the Medical Services Division is to refer such claims to the Advisory Committee on Occupational Chest Diseases unless, in the opinion of the physician subject to check by the Claims Review Branch, the claim is deemed frivolous.
- 13.5 The Corporate Board should increase the membership of the Advisory Committee on Occupational Chest Diseases (AC OCD) to not fewer than twelve physicians recommended by the Executive Director of the Board's Medical Services Division after consultation with appropriate university and professional bodies, and it should direct that the AC OCD be organized into not fewer than four subcommittees, each composed of three physicians including a subcommittee chairman designated by the Corporate Board.
- 13.6 The Corporate Board should issue the following procedural rules to conduct the operation of the Advisory Committee on Occupational Chest Diseases:
  - (i) Each subcommittee is to have the authority to diagnose claimants and assign claimants to classes of physical impairment.
  - (ii) The clinical examination of every claimant is to involve the full membership of the subcommittee to whom the claimant is assigned.
  - (iii) Every subcommittee report is to be signed by the full membership of the subcommittee, with any dissent recorded.
  - (iv) In the event of subcommittee dissent, the relevant claim is to be reviewed by the chairmen of the three remaining subcom-

mittees who shall then make the finding with respect to diagnosis and class of physical impairment.

- (v) The entire membership of the Advisory Committee on Occupational Chest Diseases is to review the findings of its subcommittees on a periodic basis.

- 13.7 The Corporate Board should adopt a rule whereby physicians engaged in the work of the Occupational Health Branch of the Ministry of Labour are ineligible for membership on the Advisory Committee on Occupational Chest Diseases and should take measures to ensure that the secretariat of this Committee is supplied solely by the Workers' Compensation Board.
- 13.8 The Occupational Health Branch of the Ministry of Labour and the Advisory Committee on Occupational Chest Diseases should continue to share the same x-ray and pulmonary function testing facilities, which should be accredited by an expert team of radiologists and respirologists at least every five years.
- 13.9 The Workers' Compensation Board, by directive of the Corporate Board, should promulgate an eligibility rule whereby survivors of deceased individuals who were rated by the Board as suffering Mild or Moderate impairment from asbestosis shall be entitled to benefits if death resulted from asbestosis, mesothelioma, lung, laryngeal, or gastrointestinal cancer, or *cor pulmonale*.
- 13.10 The *Workers' Compensation Act* should be amended so as to extend to claimants benefit of doubt in a manner consistent with that found in the *Pension Act* of the Parliament of Canada.
- 13.11 The Board should adopt a standardized form of communication with claimants which ensures:
  - (i) that all claimants for whom it is pertinent shall know that they have an explicit right to appeal and are informed of the procedures for launching an appeal; and
  - (ii) that all claimants whose disease is the subject of an eligibility rule shall have a copy of the pertinent eligibility rule.
- 13.12 The Workers' Compensation Board should adopt a standardized practice whereby:
  - (i) recipients of partial disability/impairment awards are informed of the rules pursuant to which their survivors may be eligible for benefits in the event of death; and

- (ii) in all instances where the Board has been unable to grant survivor benefits within one month of death, the claimant's survivors shall be notified that the matter is under adjudication, provided with the pertinent eligibility rules, and asked to bring forward any relevant information.

## Chapter 14 Rehabilitation, Outreach, and Prevention

**14.1** The sections of the Regulation Respecting Asbestos and the portions of its Code for Medical Surveillance which deal with the removal and rehabilitation of asbestos workers should be thoroughly revised so as to:

- (i) remove from examining physicians any role in determining whether asbestos workers are fit, fit with limitations, or unfit to work in an asbestos exposure;
- (ii) direct examining physicians to inform the worker and the Workers' Compensation Board of any finding that a worker may have or has a condition resulting from exposure to asbestos;
- (iii) vest an unequivocal right to refuse removal in the individual worker; and
- (iv) specify that removal entails moving the worker to a position where an asbestos control programme mandated by the Regulation does not apply.

**14.2** The Workers' Compensation Board should:

- (i) take steps to ensure that all examining physicians under the Regulation Respecting Asbestos are familiar with the terms of the Asbestos Fibre Dust Effect guideline, the manner in which it was developed, and its limitations;
- (ii) automatically set up a claim for any worker who, according to the finding of an examining physician, may have or has a condition resulting from exposure to asbestos;
- (iii) refer every such claim to the Advisory Committee on Occupational Chest Diseases (ACOCD);

- (iv) offer the option of removal and rehabilitation to workers who are confirmed, either by the ACODC or upon appeal, as exhibiting asbestosis or dust effects; and
- (v) review, in not less than ten years, and with the assistance of the Advisory Council on Industrial Disease Policy, the continuing appropriateness of offering removal and rehabilitation to asbestos workers.

**14.3** The Memorandum of Understanding which constitutes the formal description of the operating relationship between the Ministry of Labour and the Workers' Compensation Board should be amended to recognize a joint Ministry-Board responsibility for the development and implementation of outreach measures designed to promote the identification of potential claims for industrial disease compensation.

**14.4** The Ministry of Labour and the Workers' Compensation Board should attempt systematically to locate the residential addresses of all individuals who, because of previous asbestos exposure, have been examined by the mobile x-ray units of the Chest Surveillance Programme so that these individuals may be sent a card which they would be invited to present to their physicians whenever they wished. The card would inform the physician that its bearer was once under surveillance as an asbestos worker, alert the physician to be sensitive to any symptoms that might be indicative of asbestos-related disease, and invite the physician to contact the Ministry or the Board for records of his patient or other pertinent assistance.

**14.5** The Ministry of Labour and the Workers' Compensation Board should systematically seek the co-operation of organizations such as the Ontario Medical Association and the Ontario Chapter of the College of Family Physicians of Canada to encourage all practising physicians to take work histories of patients whenever possible symptoms of asbestos-related disease are suspected and to inform such patients on how to initiate compensation claims. The Ministry and the Board should encourage these organizations to spread an understanding of the state of occupational medicine among physicians by all appropriate means, including local district council meetings and professional publications.

**14.6** The Ministry of Labour and the Workers' Compensation Board, with the co-operation of the Ontario Hospital Association, should communicate to the chief executive officers of Ontario hospitals the



importance of taking steps that will promote, as a matter of hospital policy, the soliciting of occupational histories from patients who are admitted to hospital with symptoms of disease that may be asbestos-related.

- 14.7 The mandate of the Advisory Council on Industrial Disease Policy should include the role of monitoring the outreach efforts of the Ministry of Labour and the Workers' Compensation Board in an ongoing manner, assessing the relative success of these efforts, and advising on appropriate means of enhancing outreach.
- 14.8 The Workers' Compensation Board should levy against Johns-Manville Canada the maximum penalty assessment that can be made under section 91(7) of the *Workers' Compensation Act* in respect of the operation of the asbestos-cement pipe plant in Scarborough which, in 1980, was a member of rate group 137.
- 14.9 The *Workers' Compensation Act* should be amended to give the Workers' Compensation Board a statutory right of action against any employer the value of whose disease claims exceeds his contributions to the accident fund when the Board has reason to believe that the employer withheld from his employees knowledge which he possessed about the hazardous nature of a substance to which he exposed them. The amount of recovery by the Board should be statutorily fixed as twice the value of the employer's disease claims occasioned by the substance in question, one-half to be paid into the accident fund, the other half to the claimants involved.
- 14.10 In approaching the matter of any revisions in the experience rating of industrial disease, the Workers' Compensation Board should consider the financing of asbestos-related claims, until at least the turn of the century, as a special case. The Board should be sensitive to the proposition that the principle of horizontal equity speaks for greater rather than less experience rating among asbestos employers, and at a minimum does not countenance less experience rating than applies at present.
- 14.11 The Workers' Compensation Board should be sensitive to the potential of exposure charges on a selective basis, but should take notice that asbestos is likely not appropriate for such charges. Instances where such charges might appropriately be developed should be identified with the co-operation of the Ministry of Labour and the assistance of the Advisory Council on Industrial Disease Policy.

## **From: Part VII**

### **Learning from the Asbestos Experience**

## **Chapter 15 Identifying and Designating Hazardous Substances**

- 15.1** The Government of Ontario should take steps, using its established channels of federal-provincial communication, to promote the expeditious development of a national agency for hazard identification and risk assessment that would serve the needs of governments at all levels of jurisdiction. This agency might be a federal agency, an agency jointly created by the federal government and the provinces, or a university-based centre or free-standing, non-profit institution with a mandate and funding provided jointly by the federal and provincial governments.
- 15.2** The Ministries of Labour and of the Environment should, as soon as possible after the release of this Report, prepare and disseminate an information sheet which explains, in the simplest possible terms, the differences between the measurement techniques used to derive fibre counts in fixed workplaces and the measurement techniques used to derive fibre counts in building air, and of the consequent irrelevance of making direct comparisons between the numerical asbestos control limits of the Ministry of Labour and the numerical guidelines of the Ministry of the Environment.
- 15.3** The Minister of Labour should make a formal reference to the Advisory Council on Occupational Health and Occupational Safety concerning what measures, including statutory amendment if necessary, should be taken to permit the preliminary regulation of designated substances while the designated substance process is underway.



## **Part II**

# **Health Effects of Asbestos**





## Chapter 2 Asbestos and Disease

### A. Introduction

There have been few substances in our environment whose possible adverse health effects have received as much attention as asbestos. The medical and scientific literature on asbestos is voluminous and continues to expand;<sup>1</sup> the health hazards of asbestos have been the focus of widespread media coverage; professional and educational conferences have been organized solely to consider its effects; asbestos has been the subject matter of several commissions of inquiry in other jurisdictions;<sup>2</sup> various groups of asbestos workers continue to be subjected to epidemiological study for evidence of excess disease; claims for asbestos-caused injuries have produced numerous lawsuits, particularly in the United States,<sup>3</sup> and frequent resort to workers' compensation agencies in this province and elsewhere; and exposures to asbestos at least in occupational settings are controlled in most

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<sup>1</sup>For a fairly complete bibliography, see George A. Peters and Barbara J. Peters, *Sourcebook on Asbestos Diseases: Medical, Legal, and Engineering Aspects* (New York: Garland STPM Press, 1980); and Irving J. Selikoff and Douglas H.K. Lee, *Asbestos and Disease* (New York: Academic Press, 1978).

<sup>2</sup>For example: U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979); R.L. Zielhuis, rapporteur, *Public Health Risks of Exposure to Asbestos*, Report of a Working Group of Experts, prepared for the Commission of the European Communities (Oxford: Pergamon Press, 1977); and Québec, Comité d'étude sur la salubrité dans l'industrie de l'amiante, *Rapport final* (Beaudry Report), René Beaudry, J.C.P., Président (Québec: l'éditeur officiel du Québec, 1976).

<sup>3</sup>It has been estimated that by the end of 1982 more than 20,000 persons in the United States had instituted claims for damages for asbestos-related injuries. Almost all have been third-party product liability claims. See *The New York Times*, 28 January 1983, and see generally, the *Asbestos Litigation Reporter* (Edgemont, Pennsylvania: Andrews Publications, Inc., 1979-); and *Borel v. Fibreboard Paper Products Corporation* 493 F.2d 1076 (1973), cert. denied 419 U.S. 869 (1974). Such suits are basically statute-barred in Ontario by virtue of s. 8(9) of the *Workers' Compensation Act*, R.S.O. 1980, c. 539. See also, Chapter 14, Section C.1 of this Report.

jurisdictions.<sup>4</sup> Yet despite the close scrutiny to which the health effects of asbestos have been subjected, there is still much uncertainty and controversy on many of the most important issues. This is because the extensive study of the health effects of asbestos has refined the issues of concern. Certainly by the time this Commission was appointed, one no longer needed to ask whether asbestos was a health hazard. By April 1980, this had long been an established fact. By the time we commenced our inquiry, the inhalation of asbestos by workers who were exposed to it in their jobs had already left a tragic legacy of serious disease and death in many parts of the world.

But this legacy was the result of very high exposure levels many years ago. What is the health risk to workers at current, much lower levels of exposure? Are some types of asbestos more hazardous than others? Do some types of individual processes utilizing asbestos carry with them a greater risk of disease than others? Are the dimensions of the fibres important determinants of disease, and do they vary among individual processes? Do short yet intense exposures to asbestos of the kind to which construction, demolition, and building maintenance workers are likely exposed cause a disproportionate health risk? What is the health risk to children exposed to asbestos in schools; and, more generally, what is the health risk to all those exposed to asbestos outside the workplace? These were among the most difficult and salient questions confronting us as we began our deliberations.

In this chapter and the three that follow it, we endeavour to respond to these questions by examining the health effects of asbestos in detail. Chapter 2 provides a framework for this examination. It is devoted in the main to what is already known about asbestos — a description of what it is and its various types; of the types of processes in which it has been used; and of the diseases we know it can cause. In Chapter 3 we focus on the health effects of asbestos in this province and provide a brief history of the manner in which asbestos has been regulated in Ontario workplaces. In Chapter 4 we examine more intensively the sources of our knowledge concerning the health effects of asbestos and the criteria by which the medical and scientific evidence ought to be evaluated. Finally, in Chapter 5 we consider what we discern to be the most critical questions concerning the health effects of asbestos, weigh and assess the relevant evidence on these questions, and offer our best judgement as to the answers. In doing so we have been greatly assisted not only by a review of the considerable volume of medical and scientific literature which has been brought to our attention, but also by the sworn testimony of many of the internationally acknowl-

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<sup>4</sup>See Chapter 7, Section D of this Report.

edged experts on asbestos-related diseases,<sup>5</sup> testimony which was subjected to thorough cross-examination by the parties-with-standing at formal hearings of this Commission.

## B. What is “Asbestos”?

### B.1 General Characteristics

Asbestos is not one mineral but a generic term used to describe a family of naturally occurring fibrous hydrated silicates divided on the basis of mineralogical features into two groups: serpentines and amphiboles. The important property of asbestos as compared to non-asbestiform varieties of silicates is the presence of mineralogically long, thin fibres that can be easily separated.<sup>6</sup> According to some definitions, there are as many as thirty varieties of asbestos, but only six are of commercial importance.<sup>7</sup> These, together with their chemical composition, are depicted in Figure 2.1.<sup>8</sup> Chrysotile, which is by far the most abundant, is the only type that belongs to the serpentine group. Crocidolite and amosite, the two other most commonly used fibres, together with anthophyllite, tremolite, and actinolite belong to the amphibole group.<sup>9</sup>

The word “asbestos” is derived from the Greek word meaning “inextinguishable” and the origin of its name reflects one of its principal characteristics: fire resistance.<sup>10</sup> But asbestos has many other qualities which enhance its commercial utility, among them tensile strength, durability, flexibility, and resistance to heat, wear, and corrosion.

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<sup>5</sup>For a list of the witnesses who gave evidence before this Commission, see Appendix B to this Report.

<sup>6</sup>Eric J. Chatfield, “The Problems of Measurement of Asbestos,” in Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos, Second Public Meeting, Friday, December 12, 1980*, reported by Lydia Dotto (Toronto: Royal Commission on Asbestos, 1981), Appendix A, p. 1. The asbestoses all have non-fibrous polymorphs of similar composition. See also, Gyan S. Rajhans and John L. Sullivan, *Asbestos Sampling and Analysis* (Ann Arbor, Michigan: Ann Arbor Science Publishers, Inc., 1981), p. 5.

<sup>7</sup>See Malcolm Ross, “The ‘Asbestos’ Minerals: Definitions, Description, Modes of Formation, Physical and Chemical Properties, and Health Risk to the Mining Community,” in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, Gaithersburg, Maryland: 18–20 July 1977, NBS Special Publication 506 (Washington, D.C.: U.S. National Bureau of Standards, November 1978), pp. 49–63.

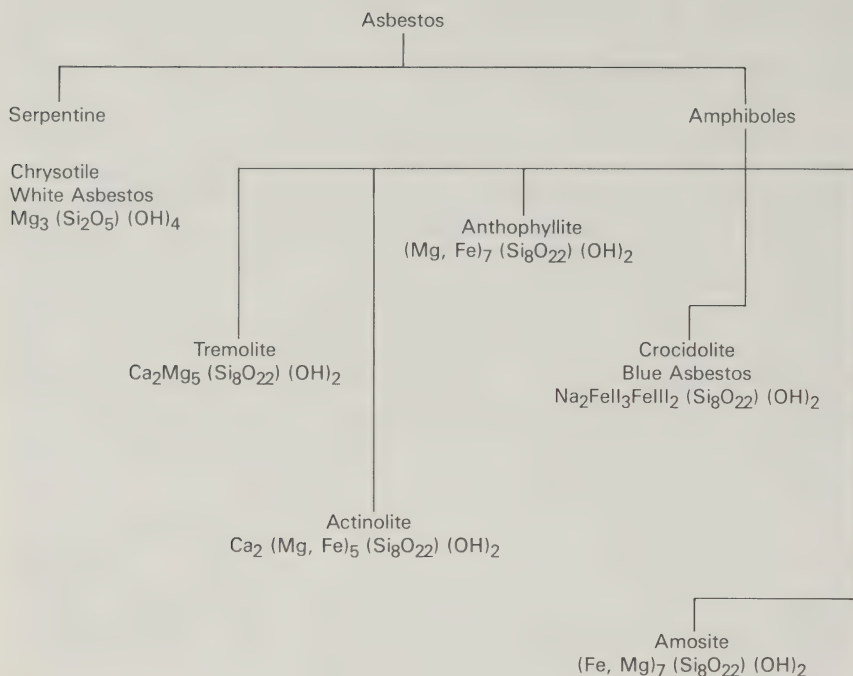
<sup>8</sup>Chatfield, “The Problems of Measurement of Asbestos,” Appendix A, Figure 1, p. 2 and accompanying text, pp. 1–4.

<sup>9</sup>World Health Organization, International Agency for Research on Cancer Working Group, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, Lyon, France: 14–17 December 1976 (Lyon: IARC, 1977), pp. 11ff.

<sup>10</sup>Jack Zussman, “The Mineralogy of Asbestos,” in *Asbestos: Properties, Applications, and Hazards*, vol. 1, eds. L. Michaels and S.S. Chissick (Chichester: John Wiley & Sons, 1979), chap. 2, p. 45.



Figure 2.1  
Principal Varieties of Asbestos



SOURCE: Dr. Eric J. Chatfield, "The Problems of Measurement of Asbestos," in Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos, Second Public Meeting, Friday, December 12, 1980*, reported by Lydia Dotto (Toronto: Royal Commission on Asbestos, 1981), Appendix A, Figure 1, p. 2.

We are aware that the mineralogical classification of what is and what is not asbestos is complex, and as a result many different definitions of asbestos have appeared in the scientific literature. We are also aware that there are non-fibrous forms of minerals with essentially the same chemical composition as the asbestos varieties. In some cases the non-fibrous form has the same name as its fibrous counterpart, for example, tremolite.<sup>11</sup> The six varieties we have listed are deemed to be asbestos only when they have a fibrous form.<sup>12</sup> In addition to these six varieties, there are many minerals with fibrous crystal habits that occur naturally, which some authorities have included in the definition of asbestos. But these latter minerals either do not possess the useful characteristics generally attributed to asbestos or they do not occur in sufficient concentrations to be commercially exploited.<sup>13</sup> Having regard to our mandate “to investigate all matters relating to health and safety arising from the use of asbestos in Ontario,” our use of the term “asbestos” will encompass those six asbestiform or fibrous minerals — chrysotile, crocidolite, amosite, anthophyllite, tremolite, and actinolite — which individuals are likely to come into contact with by reason of their commercial utility or natural occurrence.<sup>14</sup> Figure 2.2 shows the six types of asbestos pictorially. Table 2.1 sets out in tabular form the composition, properties, sources, and usage of these six fibre types.

## B.2 Chrysotile

Chrysotile is a member of the serpentine group of minerals which also includes the minerals antigorite and lizardite. However, of these three serpentine minerals, only chrysotile occurs in an asbestiform or fibrous form that may be called asbestos. Chrysotile can also occur in a non-asbestos form that is chemically and structurally similar to the chrysotile asbestos form. The main difference between them is that the chrysotile asbestos form has crystallized as parallel fibres that can be easily separated into individual fibres or fibre bundles, whereas the non-asbestos form has

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<sup>11</sup>Ross, “The ‘Asbestos’ Minerals: Definitions, Description, Modes of Formation, Physical and Chemical Properties, and Health Risk to the Mining Community”; and Chatfield, “The Problems of Measurement of Asbestos,” Appendix A, pp. 1–3.

<sup>12</sup>The Ontario Regulation Respecting Asbestos defines “asbestos” as “. . . any of the following fibrous silicates: actinolite, amosite, anthophyllite, chrysotile, crocidolite or tremolite.” See Regulation Respecting Asbestos, O. Reg. 570/82, s. 1(a), made under the *Occupational Health and Safety Act*, R.S.O. 1980, c. 321.

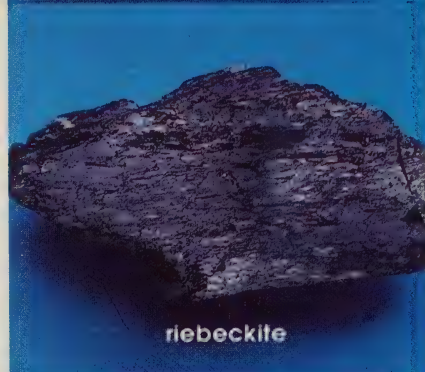
<sup>13</sup>World Health Organization, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, p. 11.

<sup>14</sup>The U.K. Advisory Committee on Asbestos took the same approach in its final report, as have the U.S. regulatory agencies. See U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee*, pp. 11–12; and Ross, “The ‘Asbestos’ Minerals: Definitions, Description, Modes of Formation, Physical and Chemical Properties, and Health Risk to the Mining Community,” pp. 49–53.

Figure 2.2  
Types of Asbestos

asbestiform

non-asbestiform



SOURCE: R.T. Vanderbilt Company, Inc., Norwalk, Connecticut. Submitted for comment by the Royal Commission on Asbestos to Mr. Fred J. Wicks, Department of Mineralogy, Royal Ontario Museum, Toronto, Ontario, August 1983.



asbestiform



non-asbestiform

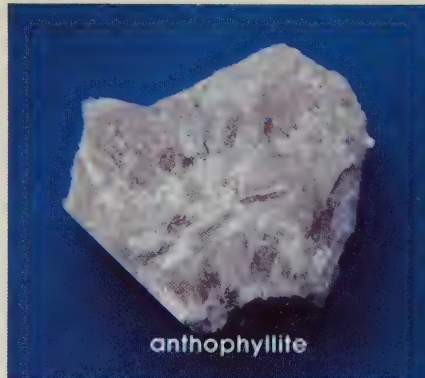




Table 2.1  
Types of Asbestos: Composition, Properties, Sources, and Usage

Variety Property	Amphiboles				
	Serpentine	Crocidolite (Riebeckite)	Amosite (Grunerite)	Anthophyllite	Tremolite
Essential composition	Chrysotile Hydrous silicate of magnesium	Silicate of Na and Fe with some water	Silicate of Fe and Mg; higher iron than anthophyllite	Mg silicate with iron	Ca and Mg silicate with water
Crystal structure	Fibrous and asbestiform	Fibrous	Prismatic, lamellar to fibrous	Prismatic, lamellar to fibrous	Long and thin columnar to fibrous
Mineralogical structure	In veins of serpentine, etc.	Fibrous in iron formation	Lamellar, coarse to fine fibrous and asbestiform	Lamellar, fibrous asbestiform	Long, prismatic, and fibrous aggregates
Mineral association	In altered peridotite adjacent to serpentine and limestone near contact with basic igneous rocks	Iron-rich silicious argillite in quartzose	In crystalline schists, iron formation	In crystalline schists and gneisses	In Mg limestones as alteration product of magnesium rocks, metamorphic and igneous rocks
Colour	Usually white to green, grey, yellow, pink	Blue	Grey, yellow to dark brown	Yellowish-brown, greyish, white	Grey-white, greenish, yellowish, bluish
Texture	Fine, soft to harsh, also silky	Soft to harsh, but flexible	Harsh and coarse but somewhat pliable	Harsh; soft to friable	Harsh or friable
					Greenish
					Harsh, brittle

Table 2.1 (continued)

Types of Asbestos: Composition, Properties, Sources, and Usage

Variety Property	Serpentine	Amphiboles			
	Chrysotile	Crocidolite (Riebeckite)	Amosite (Grunerite)	Anthophyllite	Tremolite
Lustre	Silky	Silky to dull	Vitreous to pearly	Vitreous to pearly	Silky
Hardness	2.5-4.0	4	5.5-6.0	5.5-6.0	5.5
Flexibility	High	Good	Good	Poor	Poor
Resistance to acids	Undergoes fairly rapid attack	Good	Attacked slowly	Very good	Very good
Resistance to alkalis	Very good	Good	Good	Very good	Good
Spinnability	Very good	Fair	Fair	Poor	Poor
Tensile strength lb./in. <sup>2</sup>	80,000- 100,000; 824,000 (max.)	100,000- 300,000; 876,000 (max.)	16,000- 90,000	4,000 and less	1,000- 8,000
Filtration properties	Slow	Fast	Fast	Medium	Medium
Decomposition temperature (0°C)	450-700	400-600	600-800	600-850	950-1,040
					620-960



crystallized as a complex tangle that cannot be separated into individual fibres or fibre bundles.<sup>15</sup>

Chrysotile differs from the amphiboles both structurally and in terms of chemistry. Chrysotile asbestos is composed generally of pliable, curly fibres made up of tiny individual fibrils which take the shape of a spirally wound tube.<sup>16</sup> It is the cylindrical structure of chrysotile that produces its fibrous character. Mineralogically, chrysotile is a layered silicate rolled up into a scroll and chrysotile asbestos is a number of these scrolls arranged parallel to each other. The curliness of the chrysotile fibre means that aerodynamically it behaves as if it were much thicker than its true diameter would suggest and hence neither easily becomes airborne nor efficiently penetrates through lung tissue in comparison to amphibole fibres. Chrysotile fibres have the ability to come apart very readily, splitting up into their tiny individual fibrils.<sup>17</sup>

The texture of chrysotile varies from soft, silky fibres to relatively harsh sticules. Although termed "white asbestos," chrysotile fibres may be green, grey, amber, pink, or white in colour. They are characterized by high tensile strength, a high resistance to alkalis, and, except for the harsh sticules, high flexibility and good spinnability.<sup>18</sup>

It is a common misconception that asbestos minerals are indestructible. In fact, all are susceptible to both chemical and thermal degradation. Chrysotile, when immersed in acidic fluid, loses its magnesium component, which can lead to the degradation of the fibres. Resistance to attack by other agents is in general excellent at temperatures below 100° centigrade,

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<sup>15</sup>Letter from Mr. Fred J. Wicks, Department of Mineralogy, Royal Ontario Museum, Toronto, Ontario to the Royal Commission on Asbestos, 8 September 1983.

<sup>16</sup>See Peters and Peters, *Sourcebook on Asbestos Diseases: Medical, Legal, and Engineering Aspects*, p. A2; Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 58; and RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 9.

<sup>17</sup>RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 52-54; RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 96; Victor Timbrell, "The Inhalation of Fibrous Dusts," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 255-273; Victor Timbrell, "The Inhalation of Fibres," in *Pneumoconiosis: Proceedings of the International Conference, Johannesburg, 1969*, ed. H.A. Shapiro (Cape Town: Oxford University Press, 1970), pp. 3-9; and Paul Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, p. 136.

<sup>18</sup>Zielhuis, *Public Health Risks of Exposure to Asbestos*, pp. 20-23; Eric J. Chatfield, "Measurement of Asbestos Fibres in the Workplace and in the General Environment," in Mineralogical Association of Canada, Université Laval, Québec: 20-22 May 1979, *Short Course in Mineralogical Techniques of Asbestos Determination*, ed. R.L. Ledoux (Toronto: Mineralogical Association of Canada, Royal Ontario Museum, May 1979), p. 116; and RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 61-62.



but this resistance deteriorates rapidly at higher temperatures. At temperatures of approximately 575° centigrade, the fibrous nature of chrysotile is destroyed, leaving inert particles.<sup>19</sup>

### B.3 The Amphiboles

The amphibole group of minerals is composed of a large number of structurally similar but chemically different minerals, some of which occur in both fibrous and non-fibrous forms. One of the main features of the crystal structure of the amphiboles is their distinctive cleavage that produces elongated fragments when crystals are broken. Although the direction of fibre growth is parallel to the cleavage planes in amphiboles, the fibrous character is not produced by the cleavage. Amphibole asbestos fibres appear to be produced by specific geological environments that promote the development of fibres.<sup>20</sup>

Whereas chrysotile asbestos fibres are curly in shape, the amphibole asbestos fibres are straight and needle-like. Whereas chrysotile asbestos has a spirally wound, or concentric, layered silicate structure, the amphiboles have a chain-like silicate structure. Fibres of the amphibole series are generally more brittle and tend to cleave, that is, to split longitudinally much more readily than chrysotile. The amphiboles also appear to be more dusty than chrysotile, having a greater tendency to become airborne because of their straight, dart-like characteristics.<sup>21</sup> As a group, they are more resistant to acids than chrysotile and, save for crocidolite, more resistant to heat.

As we have already stated, there are five main types of amphibole asbestos — crocidolite, amosite, anthophyllite, tremolite, and actinolite — of which crocidolite and amosite have been commercially by far the most important.<sup>22</sup>

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<sup>19</sup>RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 55–56.

<sup>20</sup>Mr. Fred J. Wicks of the Department of Mineralogy, Royal Ontario Museum has suggested that there are more than 50 minerals that make up the amphibole group. Letter from Mr. Fred J. Wicks to the Royal Commission on Asbestos, 8 September 1983.

<sup>21</sup>RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 52–53; and Rajhans and Sullivan, *Asbestos Sampling and Analysis*, pp. 8–9.

<sup>22</sup>Mr. Fred J. Wicks of the Department of Mineralogy, Royal Ontario Museum has suggested that there is a sixth member of the amphibole group found in a fibrous form in a large enough amount to be mined as asbestos: namely, potassium winchite asbestos recently found in Texas. Letter from Mr. Fred J. Wicks to the Royal Commission on Asbestos, 8 September 1983.

Crocidolite, or “blue asbestos,” is the fibrous variety of the mineral species riebeckite.<sup>23</sup> It has excellent acid-resistant properties and very high tensile strength, but only fair spinnability and poor resistance to heat.<sup>24</sup>

Amosite, which is not in fact a mineral name but an acronym for Asbestos Mines of South Africa, is mineralogically of the grunerite series.<sup>25</sup> Although known as “brown asbestos,” it in fact varies in colour from yellow to grey to dark brown. It has a coarse texture, is highly resistant to heat, and is quite flexible, but may be susceptible to strong acids and alkalis. Its tensile strength is much less than that of chrysotile or crocidolite, and it has only fair spinnability.<sup>26</sup>

Anthophyllite fibres are yellowish-brown, greyish, or white in colour. They have a harsh texture, poor flexibility, poor spinnability, and low tensile strength, but a high resistance to acids and heat.

Tremolite varies in colour from grey-white to greenish-yellow or bluish. These fibres are quite resistant to acids and heat, but have poor flexibility and spinnability. Tremolite has a silky lustre and generally a harsh texture.<sup>27</sup>

Actinolite fibres have a greenish colour, a silky lustre, a harsh texture, and may be quite hard. However, they have a comparatively low tensile strength, poor flexibility, and poor spinnability.

The poor flexibility and spinnability of anthophyllite, tremolite, and actinolite are important reasons why there have been no major industrial uses of these fibre types.<sup>28</sup>

## B.4 Commercial Deposits

Of the six fibre types, chrysotile dominates the world's commercial deposits of asbestos; 1980 production figures indicate that chrysotile alone

<sup>23</sup>Ross, “The ‘Asbestos’ Minerals: Definitions, Description, Modes of Formation, Physical and Chemical Properties, and Health Risk to the Mining Community,” p. 50.

<sup>24</sup>See Table 2.1 in this chapter and the sources referred to therein.

<sup>25</sup>Chatfield, “The Problems of Measurement of Asbestos,” Appendix A, p. 1.

<sup>26</sup>See Table 2.1 in this chapter and the sources referred to therein.

<sup>27</sup>Peters and Peters, *Sourcebook on Asbestos Diseases: Medical, Legal, and Engineering Aspects*, pp. A2–A4; Margaret R. Becklake, “Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice,” *American Review of Respiratory Disease* 114:1 (July 1976): 191; and Rajhans and Sullivan, *Asbestos Sampling and Analysis*, pp. 15–18. See also, Table 2.1 in this chapter.

<sup>28</sup>It is also true that there are few commercially exploitable deposits of these fibre types. See, for example, Zielhuis, *Public Health Risks of Exposure to Asbestos*, p. 18.

accounted for approximately 90% of all asbestos produced in the world.<sup>29</sup> The major chrysotile deposits are found in the Soviet Union and in Canada, with the principal Canadian deposits being located in the Eastern Townships of Quebec and with significantly lesser deposits in Cassiar, British Columbia; Baie Verte, Newfoundland; Clinton Creek, Yukon; and in Northern Ontario. The deposits in Newfoundland and the Yukon are not being mined at the present time.<sup>30</sup> In the recent past there were four chrysotile mines operating in Northern Ontario, but only one is still in operation.<sup>31</sup> Munro Mines in Matheson, near Timmins, suspended operations in 1968, as did Reeves Mines in Reeves Township, also near Timmins, in 1975; and Matachewan Mines outside Kirkland Lake ceased production in 1977. Hedman Mines, a small operation with less than 20 employees located near Matheson, remains open but apparently mines asbestos only sporadically.<sup>32</sup>

In addition to the Soviet Union and Canada, chrysotile is or has been mined in South Africa, Zimbabwe, Cyprus, Italy, the United States, China, Swaziland, and Brazil.<sup>33</sup>

The commercial production of amphibole asbestos is now almost completely confined to South Africa. Crocidolite is mined in the Cape and Transvaal regions of South Africa and to a very small extent in Bolivia. Australia has discontinued production of its crocidolite deposits, and a further deposit in Lusaka in Zambia has not been mined. Crocidolite, as of 1980, accounted for approximately 6% of the world's production of asbestos. Amosite is mined principally in the Transvaal and to a lesser extent in India and, as of 1980, accounted for approximately 3% of world production.<sup>34</sup>

Small amounts of anthophyllite were mined in Finland up to 1975 and are currently mined in South Africa, Bulgaria, and the United States. Fibrous actinolite is rare, but there is a small production from Taiwan and South Africa. Limited amounts of tremolite are mined in South Africa, Italy, Pakistan, and Korea.<sup>35</sup>

<sup>29</sup>A.A. Hodgson, "Chemistry and Physics of Asbestos," in *Asbestos: Properties, Applications, and Hazards*, chap. 3, pp. 68-73; and G. Oliver Vagt, *Asbestos* (Hull, Quebec: Supply and Services Canada, 1980), p. 7.

<sup>30</sup>Roskill Information Services Ltd., *The Economics of Asbestos*, 4th ed. (London, England: Roskill Information Services Ltd., 1983), pp. 8-9, 21-23. The mine at Clinton Creek was closed in 1978 and the Advocate Mines in Baie Verte were closed in 1982.

<sup>31</sup>For a more detailed description of mining and milling in Ontario, see Chapter 6, Section F of this Report.

<sup>32</sup>Ontario Federation of Labour, Written submission to the Royal Commission on Asbestos, #35, January 1981, p. 50. See also, Chapter 6, Section F of this Report.

<sup>33</sup>Roskill Information Services Ltd., *The Economics of Asbestos*, pp. 8-10.

<sup>34</sup>Vagt, *Asbestos*, p. 7; and Roskill Information Services Ltd., *The Economics of Asbestos*.

<sup>35</sup>Hodgson, "Chemistry and Physics of Asbestos," pp. 68-73.



The commercial types of asbestos are not always mineralogically pure. One type of asbestos may be contaminated by another (for example, Quebec chrysotile is contaminated by deposits of tremolite). Alternatively, one type of asbestos may contaminate a non-asbestos mineral (for example, asbestos fibres have been found in a talc mine near Madoc, Ontario; tremolite and chrysotile are found in some talc deposits in the United States; and actinolite occurs in some deposits of vermiculite).<sup>36</sup>

## B.5 Naturally Occurring Deposits

Apart from deposits that permit commercial exploitation, asbestos is ubiquitous in the crust of the earth and its surface waters. Serpentine minerals are found in the rocks of every major mountain chain in the world and in pre-Cambrian shield areas. Although concentrations large enough to mine are rare, these rocks will almost certainly contain some chrysotile asbestos. Thus, naturally occurring chrysotile will be found in soils, streams, lakes, and oceans in many localities.<sup>37</sup> The amphibole minerals also occur in a wide variety of rocks throughout the world, but most are in non-fibrous form.<sup>38</sup> The asbestiform amphiboles have a much more restricted occurrence.<sup>39</sup>

In summary, then, the deposits of asbestos can be divided into three categories: (i) deposits which are themselves commercially exploitable; (ii) deposits occurring in the rocks of mines developed primarily for the exploitation of other minerals; and (iii) natural occurrences found in many geological formations throughout the world.

## C. The Commercial Production, Processing, and Uses of Asbestos

Reports of man's earliest use of asbestos date to the Stone Age, when it was utilized to strengthen clay pottery.<sup>40</sup> From that time forward, asbestos

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<sup>36</sup>Chatfield, "The Problems of Measurement of Asbestos," Appendix A, p. 3; RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, p. 6; and Richard A. Lemen, moderator, "Discussion of Session V: Talc Contaminated with Other Minerals," in *Dusts and Disease*, eds. Richard A. Lemen and John M. Dement (Park Forest South, Illinois: Pathotox Publishers, Inc., 1979), pp. 341-355.

<sup>37</sup>RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 114-115.

<sup>38</sup>For example, there is a deposit of non-asbestiform tremolite near Sharbot Lake, Ontario. See Chapter 6, Section F of this Report.

<sup>39</sup>Fred J. Wicks, "Mineralogy, Chemistry and Crystallography of Chrysotile Asbestos," in Mineralogical Association of Canada, *Short Course in Mineralogical Techniques of Asbestos Determination*, p. 36; and Zussman, "The Mineralogy of Asbestos," p. 56.

<sup>40</sup>U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee*, paragraph 3, p. 11.



was used intermittently and in small amounts until the latter part of the nineteenth century. Canada's first asbestos mine opened in 1877 in Quebec, and the subsequent exploitation of these chrysotile deposits marked the commencement of large-scale asbestos mining and commercial production in that province.<sup>41</sup>

During the next fifty years, there was a gradual increase in world production and use, with a cumulative total of just under 5 million tons having been mined by 1930. In the years following 1930, and particularly during and after World War II, world production accelerated dramatically, nearly doubling between 1960 and 1973 alone.<sup>42</sup> This huge increase was largely attributable to the extensive use of asbestos as a fireproofing and insulation material in ship construction during the war and in building construction from the 1950s until the early 1970s. Although in recent years production has levelled off as a result of increasing concern over health effects, discontinuation of certain uses of asbestos, and the development of effective substitutes, annual world asbestos production reached over 5 million metric tons by 1979.<sup>43</sup>

Canada was once the world's leading producer of asbestos, reaching its zenith in 1947 when it mined and milled two-thirds of the world's supply. Since that time, Canada's share of world production has decreased. In general, Canadian production levels have not changed significantly since 1965, and by 1975, Canada had been overtaken by the Soviet Union. Today, Canada remains the world's second largest producer of asbestos, supplying between 25 and 30% of world production. Figure 2.3 provides a breakdown of world asbestos production by country in 1979. Table 2.2 lists asbestos production for the three principal producing countries — Canada, South Africa, and the U.S.S.R. — for the period from 1948 to 1981.

Nearly 90% of Canadian production comes from Quebec and asbestos accounts for about one-third of the value of all mineral production in the province.<sup>44</sup> Ninety-five percent of total Canadian production is exported, making it the world's leading exporter. In 1978, Canadian exports amounted to over 1.4 million metric tons, having a value in excess of \$600

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<sup>41</sup>Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 187.

<sup>42</sup>World Health Organization, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, pp. 29ff.

<sup>43</sup>Vagt, *Asbestos*, p. 7. According to *The Economics of Asbestos*, world consumption of asbestos increased from 4,231,000 metric tons in 1974 to 4,803,000 metric tons in 1980. However, in that period consumption fell 24% in North and Central America and 27% in Western Europe, while it rose 50% in South America, 86% in Eastern Europe, and 150% in Africa. Roskill Information Services Ltd., *The Economics of Asbestos*, p. 80.

<sup>44</sup>Stephen Shugar, *Effects of Asbestos in the Canadian Environment*, prepared for the NRC Associate Committee on Scientific Criteria for Environmental Quality, NRCC, no. 16452 (Ottawa: National Research Council Canada, 1979), p. 25.

million. The United States accounts for nearly 40% of total Canadian exports, and in turn, Canada supplies 99% of total United States imports of asbestos.<sup>45</sup>

Over 3,000 separate uses of asbestos have been identified in the literature. Of course, these uses depend principally upon the physical and chemical properties of asbestos fibres and the particular characteristics of individual fibre types. The following catalogue is illustrative only of the broad range of uses to which the properties of asbestos have enabled it to be put.<sup>46</sup>

*Fire retardant* — Asbestos is perhaps best known for its fire-resistant properties, and it has, for example, been widely utilized in the textile industry to increase resistance to heat and fire. Chrysotile in particular has been extensively used in textiles because its flexible and longer fibres can easily be carded, spun, and woven into yarn. Chrysotile asbestos also has been used to provide fire resistance in a wide variety of paper products.

*Insulation* — Asbestos is an effective insulator against heat, cold, electricity, and noise. Amosite and chrysotile in combination and, to a lesser extent, crocidolite were widely used in North America and elsewhere in sprayed insulation and fireproofing materials, especially in the shipbuilding industry during World War II and in the construction industry in the period immediately following the war until the early 1970s.

The heat-resistant qualities of amosite especially qualified it for these uses. The utilization of asbestos in ship and building insulation largely accounted for the huge increase in the demand for asbestos that began during the war. Thus, for example, asbestos was used as a thermal insulator in pipes and boilers, in the structural steelwork of highrise buildings, and also for acoustical and decorative purposes in ceiling tiles and building walls.<sup>47</sup> But these uses carried with them a serious health risk. The North American insulators, working with amosite and chrysotile asbestos, have one of the worst disease records of any group of employees that has been exposed to asbestos, a record first reported on in 1964 by Dr. Irving J. Selikoff and his

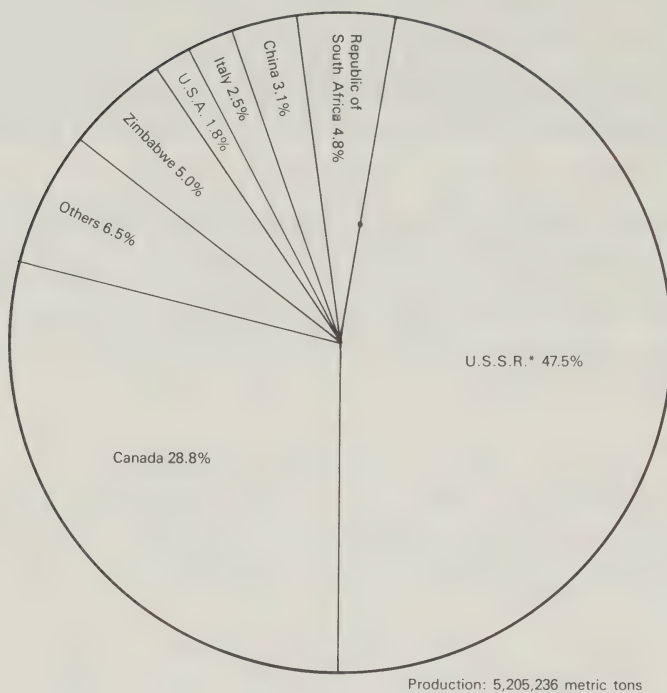
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<sup>45</sup>Gouvernement du Québec, Ministère de l'Énergie et des Ressources, Written submission to the Royal Commission on Asbestos, #19, January 1981, p. 4.

<sup>46</sup>See generally, Rajhans and Sullivan, *Asbestos Sampling and Analysis*, pp. 24ff.; Peters and Peters, *Sourcebook on Asbestos Diseases: Medical, Legal, and Engineering Aspects*, pp. A4-A7; and Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, *passim*.

<sup>47</sup>Irving J. Selikoff, "Asbestos-Associated Disease," in *Public Health and Preventive Medicine*, 11th ed., ed. John M. Last (New York: Appleton-Century-Crofts, 1980), chap. 13, p. 585.

Figure 2.3  
World Asbestos Production by Country, 1979



**Note:** \* Approximately equivalent to Canadian grades.

**SOURCE:** G. Oliver Vagt, *Asbestos* (Hull, Quebec: Supply and Services Canada, 1980), Figure 2, p. 7 (based on numerous sources).

**Table 2.2**  
**World Production of Asbestos, by Principal Producing Countries:**  
**1948 to 1981 (000 metric tons)**

Year	Canada		South Africa		U.S.S.R.		World Total
	000t	%	000t	%	000t	%	000t
1948	590	63	38	4	-	-	-
1949	473	53	58	7	-	-	-
1950	720	61	72	6	-	-	-
1951	801	62	88	7	-	-	-
1952	765	59	110	9	-	-	-
1953	827	58	86	6	272	19	1,420
1954	838	55	99	7	304	23	1,515
1955	965	55	109	6	408	23	1,755
1956	920	52	124	7	434	25	1,970
1957	949	50	143	8	434	24	1,887
1958	839	45	159	9	499	27	2,055
1959	953	46	165	8	544	26	2,059
1960	1,015	46	160	7	599	27	2,214
1961	1,065	42	177	7	798	32	2,513
1962	1,103	40	201	7	998	36	2,742
1963	1,157	46	187	8	685	27	2,505
1964	1,288	47	196	7	735	27	2,768
1965	1,259	44	218	8	785	28	3,146
1966	1,342	44	251	8	839	28	3,047
1967	1,317	45	244	8	769	26	2,910
1968	1,370	46	236	8	800	27	2,985
1969	1,431	44	258	8	962	30	3,265
1970	1,507	43	287	8	1,066	31	2,490
1971	1,483	41	322	9	1,152	32	3,585
1972	1,530	41	323	9	1,220	32	3,774
1973	1,690	43	334	8	1,280	31	4,179
1974	1,644	38	333	8	1,360	32	4,274
1975	1,056	26	355	9	1,650	41	3,992
1976	1,536	32	367	8	1,850	39	4,787
1977	1,517	32	380	8	1,900	40	4,793
1978	1,422	30	257	5	1,945	42	4,673
1979	1,493	30	249	5	2,100	42	4,992
1980	1,323	27	277	6	2,250	45	4,988
1981	1,133	24	273	6	2,340	49	4,817

SOURCE: Roskill Information Services Ltd., *The Economics of Asbestos*, 4th ed. (London, England: Roskill Information Services Ltd., 1983), Table 6, p. 9.



colleagues at Mount Sinai Hospital in New York.<sup>48</sup> Moreover, the asbestos-sprayed materials themselves flaked from building pipes and tiles with constant wear. Consequently, building occupant exposure to asbestos, however minimal, became a concern. Sprayed insulation materials were effectively banned in the United States in 1973 and their use was discontinued in Ontario in the same year.<sup>49</sup>

**Friction and Wear** — Because of its heat resistance and other qualities, chrysotile is extensively used in friction products, including linings for disc and drum brakes and clutch facings.

**Reinforcement** — Chrysotile, because of its very high tensile strength, resistance to alkalis, and flexibility, is used as a reinforcing agent in the asbestos-cement industry, whose products include asbestos-cement pipe, shingles, clapboards, and flat and corrugated sheets.<sup>50</sup> Because it enhances the reinforcement, the dispersion of the fibre, and the drainage properties of the asbestos-cement mix, limited amounts of crocidolite are used in this industry as well, particularly for the manufacture of asbestos-cement pressure pipe.<sup>51</sup>

**Wear Resistance** — Asbestos is employed in vinyl floor tiles providing excellent resistance to wear and tear, impermeability to water, strength, and fire resistance.

**Resistance to Acids and Alkalies** — Due to its excellent resistance to the corrosive action of acids and alkalies, crocidolite has been used mainly in the manufacture of battery boxes, packings, acid pumps, valves, and gaskets.<sup>52</sup>

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<sup>48</sup>Irving J. Selikoff, Jacob Churg, and E. Cuyler Hammond, "Asbestos Exposure and Neoplasia," *Journal of the American Medical Association* 188 (1964): 22-26. Idem, "The Occurrence of Asbestosis Among Insulation Workers in the United States," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 139-155; and Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91-116.

<sup>49</sup>The stringent control requirements prescribed by Regulation 419/73 made under *The Construction Safety Act* forced their discontinuance in Ontario. See Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, p. 16. In the United States, the Environmental Protection Agency in 1973 prohibited "visible emissions" of asbestos from any work site. This effectively abolished the spraying of asbestos materials for fireproofing or decorative purposes. See William J. Nicholson, "Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States," *Annals of the New York Academy of Sciences* 329 (26 October 1979): 293-303.

<sup>50</sup>Zielhuis, *Public Health Risks of Exposure to Asbestos*, p. 20.

<sup>51</sup>Ibid., p. 25.

<sup>52</sup>Ibid.

*Filtering* — During World War II, both crocidolite and chrysotile were used as the filter material in gas mask cannisters.<sup>53</sup> Asbestos fibres are also used today as a filter medium in the chemical, drug, and food industries.

*Cohesion* — The addition of small amounts of asbestos to asphalt-surfaced roadways has served to increase the cohesion and wear resistance of the pavement.

*Filler* — Asbestos is used as a filler in resins, plastics, caulking, and sealants.

Some of the individual processes that utilize asbestos are quite different and these differences may be significant in terms of health effects. All forms of manufacturing using asbestos are likely to involve opening bags and then emptying them into a hopper where the asbestos is mixed with other material. A good deal of manufacturing, including, for example, the manufacture of friction products, encompasses a step where the asbestos, mixed with binders and other materials, is ground up. By contrast, in textile spinning and weaving, the asbestos is formed into yarn, and the yarn is then moved at high speed over pulleys. These different processes may cause the release of fibres of different dimensions and hence differences in health effects, a subject we consider in detail in Chapter 5 of this Report.

We describe Canadian production and consumption of asbestos in Chapter 6 of this Report.

## D. Asbestos-Related Diseases

Since at least the beginning of this century, medical and scientific literature has documented the health hazards resulting from the inhalation of asbestos dust. As we began our inquiry, it was a well-accepted fact that asbestos exposure can cause and has indeed caused serious and debilitating disease and often death. In this section we consider the diseases associated with asbestos exposure, namely, asbestosis, mesothelioma, carcinoma of the lung (commonly referred to as lung cancer), and other asbestos-related cancers. Each of these diseases is characterized by a period of latency: that is, the existence of a time interval, usually of many years, between first ex-

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<sup>53</sup>Alison D. McDonald and J. Corbett McDonald, "Mesothelioma After Crocidolite Exposure During Gas Mask Manufacture," *Environmental Research* 17 (December 1978): 340-346; and J.S.P. Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 637-653.

posure to asbestos and the clinical manifestation of the disease.<sup>54</sup> Figure 2.4 depicts the principal asbestos-related diseases and conditions and their sites in the human body.

## D.1 Asbestosis

Asbestosis is a chronic, restrictive lung disease due to the inhalation of asbestos fibres. It is characterized by diffuse interstitial fibrosis or scarring which produces a "small, tight lung." The most prominent symptom is breathlessness and a characteristic physical sign is the presence of crepitations (rales) or crackling noises in the lung. Clubbing or thickening of the fingers and toes may be present in cases of advanced disease. Typical pulmonary function changes include restriction of lung volume, diminished forced vital capacity, and a decrease in diffusion capacity. Characteristic x-ray changes of asbestosis are linear and irregular small opacities in the lower and middle lung zones.<sup>55</sup>

There is an incomplete correlation between the extent of x-ray abnormality (and in some cases measured lung function defects) and clinical well-being. Some patients whose x-rays show extensive scarring are able to work without difficulty and live without restraint, and the opposite is also true. There is some evidence to indicate that lung function changes are often a better early detector of asbestosis than x-ray changes, but this is not invariably the case.<sup>56</sup>

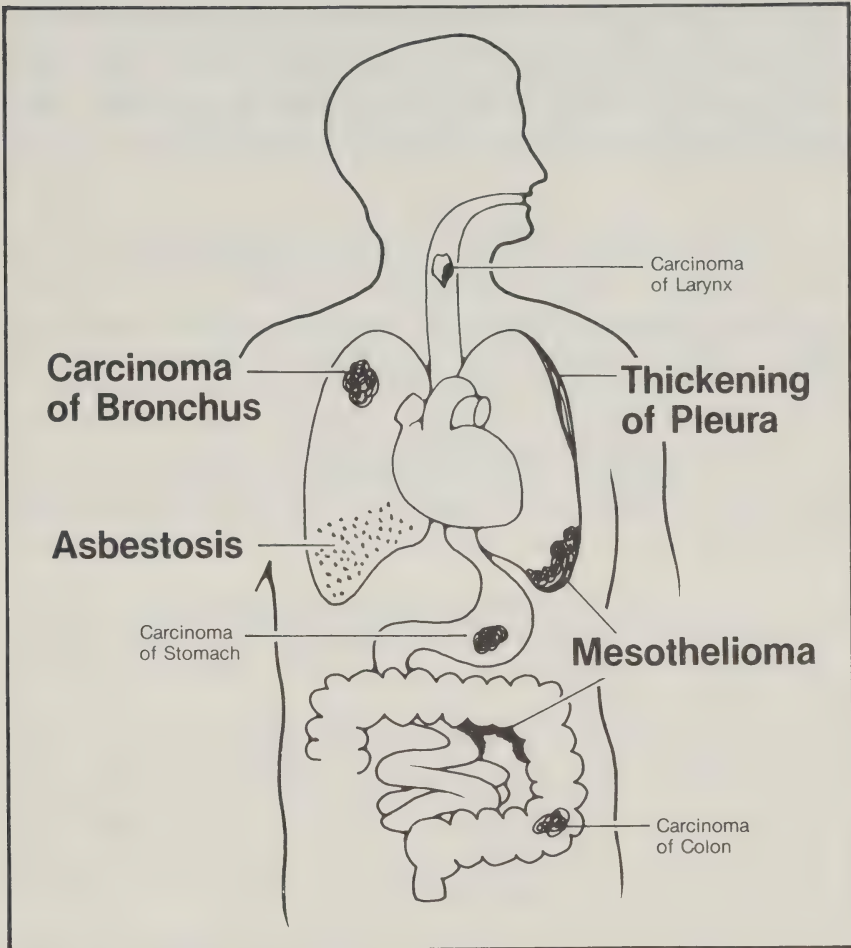
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<sup>54</sup>See generally, Selikoff and Lee, *Asbestos and Disease*; and Selikoff, "Asbestos-Associated Disease," pp. 568-598. In the brief discussion that follows, no attempt is made to distinguish among different fibre types. The question of the relative pathogenicity of the various fibre types will be left to Chapter 5 of this Report.

<sup>55</sup>Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," pp. 205-206; RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 43-44; U.S., National Institute for Occupational Safety and Health, *Revised Recommended Asbestos Standard*, prepared by Richard A. Lemen and John M. Dement (Washington, D.C.: U.S. Department of Health, Education and Welfare, December 1976), pp. 29-30; RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, p. 63; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 55-56. With silicosis, and other fibrotic lung disease, the opacities are small and rounded. Often there will be exposure to both silica and asbestos.

<sup>56</sup>Selikoff, "Asbestos-Associated Disease," pp. 571ff.; and Hans Weill et al., "Lung Function Consequences of Dust Exposure in Asbestos Cement Manufacturing Plants," *Archives of Environmental Health* 30:2 (February 1975): 95. Dr. Hans Weill examined the lung function consequences of dust exposure in two asbestos-cement manufacturing plants in the New Orleans area. His findings indicated that individuals may have a reduction in pulmonary function prior to the time when x-ray abnormalities appear.

**Figure 2.4**  
**Principal Asbestos-Related Diseases and**  
**Conditions and Their Sites in the Human Body**



SOURCE: Illustration by Mr. Jerry Farrell, Audio-Visual Centre, McMaster University; consultative assistance by Dr. David C.F. Muir, Director, Occupational Health Program, Health Sciences Centre, McMaster University, Hamilton, Ontario.



Asbestosis has historically been associated with prolonged and heavy occupational exposure to asbestos.<sup>57</sup> The pulmonary fibrotic changes develop slowly over the years, are irreversible, and will normally progress even in the absence of further exposure to asbestos.<sup>58</sup> The disease can become seriously disabling. Asbestosis is, however, more often a cause of morbidity (illness) among asbestos workers than it is a cause of mortality. Still, those with asbestosis are susceptible to death from related causes — often infection or cardio-respiratory complications. For example, pulmonary hypertension is frequently associated with advanced asbestosis and the resultant right-sided heart failure (*cor pulmonale*) may be the stated cause of death.<sup>59</sup>

There is no effective treatment for asbestosis itself, which is irreversible. However, the physician's improved ability to treat some of the symptoms and secondary effects of the fibrosis has somewhat bettered the quality of life of patients with asbestosis. Present-day medical therapy now makes it possible to give the asbestotic some relief from the discomfort associated with complications arising from asbestosis.<sup>60</sup>

It is clear that individuals afflicted with asbestosis have an appreciable reduction in their life expectancy. Mr. Geoffrey Berry calculated that persons given a 10% disability rating for asbestosis by the British Pneumoconiosis Medical Panel had a reduction in life expectancy of three years, rising to

<sup>57</sup>RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 52; RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 96; Selikoff and Lee, *Asbestos and Disease*, p. 266; and Zielhuis, *Public Health Risks of Exposure to Asbestos*, p. 78.

<sup>58</sup>RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 12–13; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 123; and Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 209. In her evidence, Dr. Margaret R. Becklake suggested that with certain fibrotic diseases there is a stage of activity at which intervention may prevent progression to fibrosis. But she thought it unlikely that it would ever be possible to identify such a stage in the asbestos-exposed individual. See RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 43–44.

<sup>59</sup>U.S., NIOSH, *Revised Recommended Asbestos Standard*, pp. 29–30; Selikoff, "Asbestos-Associated Disease," pp. 571–575; RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 69–70; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 27; RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 136; RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1981, Volume no. 49, pp. 46–47; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 42, 126–127. As Dr. Weill testified, a person with asbestosis will have less lung reserve to cope with a serious infection such as pneumonia. In addition, fairly advanced asbestosis produces changes in pulmonary circulation that in turn put a strain on the heart. Moreover, it is possible that advanced asbestosis could preclude operations in respect of other diseases.

<sup>60</sup>Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 209; RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, p. 44; RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), pp. 79–80; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 46.

twelve years for those 50% or more disabled.<sup>61</sup> In 1981, Dr. Murray M. Finkelstein, a medical consultant with the Ontario Ministry of Labour, reported on the mortality experience of 172 workers who had been awarded compensation for asbestosis by the Workers' Compensation Board. Not only did he find that these workers had increased rates of death compared to the general population for non-malignant respiratory disease, lung cancer, and mesothelioma, but he also found that in comparison with the general population, the proportion of workers who survived was only 69% five years after being awarded compensation, and only 53% after ten years.<sup>62</sup>

## D.2 Mesothelioma

Mesothelioma is a rather rare cancer arising from the surface-lining cells of the pleura and the peritoneum. These surface-lining cells are called the mesothelium and hence the name of the disease, mesothelioma. Malignant mesothelioma is of two types, pleural and peritoneal, depending upon whether the site of the tumour is the lung or the abdomen. The tumours that appear are unusual and striking. They are generally diffuse, spreading rapidly and widely over the large surfaces of the thoracic or abdominal cavities.<sup>63</sup>

There is no effective treatment for mesothelioma. Chemotherapy, immunology, radiation, and various forms of surgery have all produced disappointing results. At best, treatment programmes provide some brief measure of palliative relief or some short-term prolongation of what is certainly a very miserable life.<sup>64</sup> As Dr. Paul Kotin bluntly testified, for mesothelioma, morbidity and mortality are in practical terms identical. A large proportion of mesothelioma patients die within a year of diagnosis, and few survive longer than five years.<sup>65</sup> This bleak picture serves to emphasize that mesothelioma, like the other asbestos-related diseases, is a disease for which the prevention is far more effective than the cure.

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<sup>61</sup>Geoffrey Berry, "Mortality of Workers Certified by Pneumoconiosis Medical Panels as Having Asbestosis," *British Journal of Industrial Medicine* 38 (1981): 135; and RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, pp. 37-38.

<sup>62</sup>Murray M. Finkelstein, Robert A. Kusiak, and George Suranyi, "Mortality Among Workers Receiving Compensation for Asbestosis in Ontario," *Canadian Medical Association Journal* 125 (1 August 1981): 259-262.

<sup>63</sup>Selikoff, "Asbestos-Associated Disease," p. 580.

<sup>64</sup>Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 213; RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 135-137; and P. Ruffié and A. Hirsch, "A Review of the Treatment of Diffuse Malignant Pleural Mesothelioma," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 553-557.

<sup>65</sup>RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 81; Selikoff, "Asbestos-Associated Disease," p. 583; and Selikoff and Lee, *Asbestos and Disease*, pp. 302-306.

Historically, the incidence of mesothelioma has been extremely high among three occupational groups exposed to asbestos: insulators, those who work in asbestos plants, and those who work in shipyards. But cases of mesothelioma have been known to occur not only among those occupationally exposed to asbestos but also among persons living in the same household as an asbestos worker and among persons living in the neighbourhood of asbestos mining or manufacturing activities.<sup>66</sup> Since 1966, the incidence of mesothelioma in Canada has been on average 31 cases per year.<sup>67</sup>

Mesothelioma was for a time thought to be uniquely associated with asbestos exposure. On the basis of the available data, there is still little doubt that the great majority of reported cases of mesothelioma are attributable to asbestos exposure.<sup>68</sup> While it is true there is now a growing body of evidence which strongly suggests that mesothelioma can occur and indeed has occurred in the absence of any known or likely exposure to asbestos, these cases are, comparatively, exceedingly rare.

The evidence that mesothelioma can be caused without asbestos exposure comes principally from three sources. First, there have been a number of case reports of mesothelioma from various locales where there was simply no possibility of asbestos exposure. Chief among them is the recently reported outbreak of mesothelioma in rural Turkey where there is no industry and no asbestos. These cases have been specifically attributed to expo-

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<sup>66</sup>See J. Corbett McDonald and Alison D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," *Preventive Medicine* 6:3 (September 1977): 426-446; Selikoff and Lee, *Asbestos and Disease*, Table 10-5, pp. 267-270 and Table 11-6, p. 293; Henry A. Anderson and Irving J. Selikoff, "Asbestos-Associated Radiographic Changes Among Household Contacts of Amosite Asbestos Workers," in *Induced Disease: Drug, Irradiation, Occupation*, ed. Leslie Preger (New York: Grune and Stratton, 1979), pp. 253-273; and Muriel L. Newhouse and Hilda Thompson, "Epidemiology of Mesothelial Tumors in the London Area," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 579-588.

<sup>67</sup>J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," p. 431. Between 1966 and 1975, the annual incidence of malignant mesothelioma per million males aged 15 and over was 4.9 in Quebec, 2.6 in Ontario, and 2.1 in the rest of Canada. However, misdiagnosis, or more accurately over-diagnosis, may have been a factor: the corresponding figures for accepted incidence after approval of diagnosis by a recognized panel of Canadian pathologists were 2.4, 2.0, and 1.5 respectively. See also, Alison D. McDonald, "Mesothelioma Registries in Identifying Asbestos Hazards," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 447; and RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, p. 15. For a fuller discussion of the difficulties of diagnosing mesothelioma, see Chapter 4, Section D.2 of this Report.

<sup>68</sup>See J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," p. 439; and Alison D. McDonald and J. Corbett McDonald, "Malignant Mesothelioma in North America," *Cancer* 46:7 (1 October 1980): 1650-1656.



sure to fibrous zeolites found in the region.<sup>69</sup> Second, animal experimental work has demonstrated that glass and other man-made mineral fibres are capable of inducing mesothelioma in rats at rates which closely approximate those for asbestos.<sup>70</sup> Third, in a number of epidemiological surveys of mesothelioma, there are some cases for which there is no known history of asbestos exposure, be it occupational, domestic, or neighbourhood, and for which there is no evidence of asbestos involvement in the lung. Dr. J. Corbett McDonald and Dr. Alison D. McDonald, in their survey paper, "Epidemiology of Mesothelioma from Estimated Incidence," examined over 4,500 fatal cases of mesothelioma reported from 22 countries in the years from 1959 to 1976. In 923 cases, accounting for 38% of the cases from non-occupational studies with a recorded history, the authors were unable to find any definite or even probable history of asbestos exposure.<sup>71</sup> Similarly, Mr. Julian Peto and his colleagues, in a study of mesothelioma deaths in the Los Angeles area of California in the years from 1972 to 1979, could not establish evidence of any asbestos exposure in nearly one-third of the 188 total cases that occurred and in well over half of the female victims.<sup>72</sup> These statistics must be put in context. The numerically few cases of mesothelioma among those with no likely asbestos exposure are drawn

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<sup>69</sup>See Y. Izzettin Baris, Mustafa Artvinli, and A. Altay Sahin, "Environmental Mesothelioma in Turkey," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 423-432; and Fred D. Pooley, "Evaluation of Fibre Samples Taken from the Vicinity of Two Villages in Turkey," in *Dusts and Disease*, pp. 41-44. The data come from the villages of Karain and Tuzköy in Turkey. In the village of Karain in the years from 1970 to 1974, with a population between 600 and 800, there were a total of 24 pleural mesotheliomas. In 1974 alone, 11 of the 18 deaths in the village were from this tumour. The fibrous zeolite mineral erionite has been particularly implicated.

<sup>70</sup>And, as with asbestos, it is the longer and thinner man-made mineral fibres which appear to be more pathogenic in relation to tumour induction in animals. See Mearl F. Stanton and Constance Wrench, "Mechanisms of Mesothelioma Induction with Asbestos and Fibrous Glass," *Journal of the National Cancer Institute (JNCI)* 48:3 (March 1972): 797-821; and Mearl F. Stanton and Maxwell Layard, "The Carcinogenicity of Fibrous Minerals," in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, pp. 143-150. The issue of fibre dimension is discussed in Chapter 5, Section D of this Report.

<sup>71</sup>J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," p. 429. The authors identified 268 cases from occupational studies, all with asbestos exposure; the authors also identified 4,271 cases from other studies, of which they had a recorded history for 2,453 cases. Of these 2,453 cases, 923 had no definite or probable history of asbestos exposure.

<sup>72</sup>Julian Peto, Brian E. Henderson, and Malcolm C. Pike, "Trends in Mesothelioma Incidence in the United States and the Forecast Epidemic Due to Asbestos Exposure During World War II," in *Banbury Report 9: Quantification of Occupational Cancer*, eds. Richard Peto and Marvin Schneiderman ([Cold Spring Harbor, New York]: Cold Spring Harbor Laboratory, 1981), pp. 51-69; and RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 133.



from the entire population<sup>73</sup> and are therefore an infinitesimally small fraction of the cases of death in this population. By contrast, the much larger number of cases of mesothelioma with known or probable asbestos exposure is a noticeable fraction of a much smaller asbestos-exposed population.

Accordingly, in our view, mesothelioma remains a disease that is quite specifically linked to asbestos exposure. While we cannot conclude that asbestos exposure is the sole cause of mesothelioma and cannot discount the possibility that present and future industrial processes using non-asbestos fibres will produce mesotheliomas, we can state that the possibility that an individual without asbestos exposure will contract the disease is remote.<sup>74</sup>

### D.3 Lung Cancer

Lung cancer, unlike asbestosis or mesothelioma, is not specifically associated with asbestos exposure. Moreover, it has had a particular history of association with cigarette smoking.<sup>75</sup> Pathologically, there does not appear to be any basic difference between pulmonary carcinoma in general and that associated with persons exposed to asbestos, save that whereas the majority of tumours appear in the upper portion of the lungs, those in asbestos-exposed individuals appear more commonly (but not always) in the lower lobes.<sup>76</sup> However, serious issues of causation have been raised when lung cancer develops in asbestos workers, especially in the absence of co-existing asbestosis. While most persons exposed to asbestos who develop lung cancer are smokers, it is now generally recognized that asbestos on its own in the absence of smoking is capable of inducing lung cancer and that

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<sup>73</sup>More accurately, this means the entire non-asbestos population. The McDonald and McDonald survey included 22 countries. Before these statistics can be accepted at face value, two observations need be made: first, it is possible that the histories of those patients for whom there is said to be no asbestos exposure is inaccurate because such exposure has occurred without their knowledge or the knowledge of those interviewed; second, mesothelioma was and continues to be a difficult diagnosis and the possibility for misdiagnosis is considerable. These observations do perhaps cast some doubt on the accuracy of the mesothelioma surveys, but it is not warranted in our view to discount in their entirety the numerous reported cases with no demonstrable asbestos exposure in light of the consistency of such a finding in the various groups of cases that have been studied.

<sup>74</sup>It is in fact so rare that there is no calculable background incidence rate for mesothelioma in the general population.

<sup>75</sup>E. Cuyler Hammond, Irving J. Selikoff, and Herbert Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 473-490.

<sup>76</sup>RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 78.

the tumour may develop even where there is no co-existing asbestosis.<sup>77</sup> The relationship between asbestos, cigarette smoking, and human diseases will be explored further in Chapter 5 of this Report.

The prognosis and treatment of lung cancer in those exposed to asbestos are little different from lung cancer in the general population. Only about one in twenty persons with lung cancer survives longer than five years from the diagnosis of the disease.<sup>78</sup> If asbestosis is present, the prospects for a person with an asbestos-related lung cancer could be worse because the asbestosis may limit the treatment options available to the physician and may in particular preclude surgical intervention.<sup>79</sup>

#### D.4 Other Asbestos-Related Cancers

Other malignant neoplasms have also come to be associated with the inhalation of asbestos. An excess risk of gastrointestinal cancer affecting all sites in the gastrointestinal tract (esophagus, stomach, colon, and rectum)

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<sup>77</sup>Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 214; Selikoff and Lee, *Asbestos and Disease*, p. 309; RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 76 and 23 July 1981, Volume no. 21(B), pp. 19-20; Hammond, Selikoff, and Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," pp. 485-488; and J. Corbett McDonald, "Asbestos and Lung Cancer: Has the Case Been Proven?" *Chest* 78:2 (August 1980, Supplement): 374-376.

<sup>78</sup>RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 81; and Selikoff, "Asbestos-Associated Disease," p. 575.

<sup>79</sup>Asbestosis may limit the treatment options for other diseases as well; see, for example, the case of Mr. John Dodds, referred to in Chapter 13, Section C.1 of this Report. See Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 214.

has been demonstrated in a number of studies of asbestos workers.<sup>80</sup> More recently, certain studies have also implicated asbestos as a cause of cancer of the larynx.<sup>81</sup> However, the association between asbestos exposure and both gastrointestinal cancer and cancer of the larynx is neither as strongly nor as consistently established in the medical literature as the association between asbestos exposure and the diseases previously discussed.<sup>82</sup>

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<sup>80</sup>Selikoff, "Asbestos-Associated Disease," pp. 583-584; and Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 214. Both Dr. Alexander C. Ritchie and Dr. Anthony B. Miller reviewed the evidence pertaining to asbestos exposure and gastrointestinal cancer for the Ontario Workers' Compensation Board. Dr. Ritchie in 1976 found there was little non-epidemiological evidence to support the suggestion that exposure to asbestos increases the risk of developing carcinoma of the gastrointestinal tract. His conclusion that asbestos exposure increases the risk of developing carcinoma of the colon and rectum and probably of the esophagus and stomach rests almost entirely on the epidemiological evidence and principally on the work of Dr. Selikoff and his colleagues. Dr. Miller concluded that there is no doubt that an association has been demonstrated for all sites in the gastrointestinal tract, but that the demonstrated association is weak. See The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 27. Dr. E. Donald Acheson and Dr. Martin J. Gardner, in their report to the U.K. Advisory Committee on Asbestos, expressed reservations as to the association. In his testimony before us, Dr. Acheson was of the view that the evidence since the 1979 publication of his co-authored report of an association between asbestos exposure and gastrointestinal cancer was if anything a little weaker, and he suggested the possibility of another factor at work. See E. Donald Acheson and Martin J. Gardner, "The Ill Effects of Asbestos on Health," in U.K., Advisory Committee on Asbestos, *Asbestos — Volume 2: Final Report of the Advisory Committee*, paragraphs 211-213, p. 41; and RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, pp. 6, 72.

<sup>81</sup>P.M. Stell and T. McGill, "Asbestos and Laryngeal Cancer," *The Lancet* 2:826 (25 August 1973): 416-417; R.W. Morgan and P.T. Shettigara, "Occupational Asbestos Exposure, Smoking, and Laryngeal Carcinoma," *Annals of the New York Academy of Sciences* 271 (28 May 1976): 308-310; and M.L. Newhouse, M.M. Gregory, and H. Shannon, "Etiology of Carcinoma of the Larynx," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 687-695. Both Dr. Alexander C. Ritchie and Dr. Anthony B. Miller reviewed the evidence on asbestos exposure and cancer of the larynx for the Ontario Workers' Compensation Board. See The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, pp. 31-32, 40, 42.

<sup>82</sup>An association between asbestos exposure and other cancers, for example, cancer of the kidney, has also been suggested but not satisfactorily demonstrated. See Selikoff, "Asbestos-Associated Disease," p. 584. In their update of "The Ill Effects of Asbestos on Health," Dr. E. Donald Acheson and Dr. Martin J. Gardner suggested a possible association between asbestos exposure (likely amphiboles) and ovarian cancer in women. See E. Donald Acheson and Martin J. Gardner, *Asbestos: The Control Limit for Asbestos*, prepared for the U.K. Health and Safety Commission (London: Her Majesty's Stationery Office, 1983), paragraph 17, p. 3.

## D.5 Other Asbestos-Related Conditions

### (a) Pleural Changes (pleural thickening, pleural effusions, and pleural plaques)

Asbestos exposure can also produce scarring, both localized and diffuse, of the lining of the pleural surfaces, giving rise to pleural thickening, benign pleural effusions, and pleural plaques.<sup>83</sup> Plaques are white, glistening, raised areas of fibrous tissues which may calcify or harden. In general, these limited pleural changes and plaques are not associated with clinical and functional abnormalities. They are simply markers of asbestos exposure. However, once in a while this pleural process can become rather diffuse and can severely restrict lung function, thereby incapacitating the individual.<sup>84</sup> Pleural changes may or may not be accompanied by asbestosis. Some schools of medicine, notably the Mount Sinai School of Medicine in New York, consider these pleural changes as “pleural asbestosis,” but the term is not widely used, undoubtedly because these changes generally do not produce clinically disabling effects similar to asbestosis.<sup>85</sup>

### (b) Asbestos Bodies and Warts

Certain particles of asbestos fibres, once inhaled in the lungs, may become coated with a substance containing protein and iron to form asbestos bodies. Fibres other than asbestos can provide a core on which similar coatings can develop and therefore the generic term “ferruginous bodies” is often used. Asbestos bodies are not themselves harmful but represent a benign tissue reaction to asbestos fibres. Principally they serve as evidence

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<sup>83</sup>The plaques may be hyaline or calcified. See Becklake, “Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice,” pp. 200–202.

<sup>84</sup>RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 13–14; and RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 66.

<sup>85</sup>RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 14; and Selikoff and Lee, *Asbestos and Disease*, pp. 143ff. and especially pp. 189ff. The Mount Sinai School of Medicine in New York distinguishes between “pleural” and “parenchymal” asbestosis.



of asbestos exposure,<sup>86</sup> as do asbestos warts which are harmless skin growths that occur when asbestos fibres penetrate the skin.<sup>87</sup>

## E. The Recognition of Asbestos as a Health Hazard and the Historical Incidence of Disease

It was the fibrogenic effects of asbestos inhalation that triggered the recognition of asbestos as a health hazard. The earliest known cases of asbestosis were among asbestos textile workers. For example, in 1906, M. Auribault, an inspector in the Department of Labour in France, reported some 50 deaths in the period 1890–1895 among workers at an asbestos weaving mill in Calvados.<sup>88</sup> In 1907, Dr. H. Montague Murray related the death of a patient from fibrosis of the lungs due to the inhalation of asbestos dust to a parliamentary committee on industrial disease compensation in Great Britain, the victim being a 33 year old man who had worked for 10 years in the carding room of an asbestos textile mill.<sup>89</sup> In 1912, a Canadian health inspector who had examined conditions in an asbestos factory and in asbestos mines and mills noted that large amounts of asbestos dust had a weakening effect on the lungs.<sup>90</sup> The insurance industry too recognized the health hazards of asbestos at an early stage. Dr. Frederick L. Hoffman, statistician and Vice-President of the Prudential Insurance Company, wrote in 1918 “. . . that in the practice of American and Canadian life insurance companies asbestos workers are generally declined

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<sup>86</sup>Becklake, “Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice,” pp. 194ff.; and Canadian Centre for Occupational Health and Safety, *A Review of Four Major Reports on the Health Hazards of Asbestos*, Royal Commission on Asbestos Background Paper Series, no. 2 (Toronto: Royal Commission on Asbestos, 1981), p. 4. Asbestos bodies are found in the lungs of many urban residents. They are not harmful but are simply indicative of an environmental exposure to asbestos. It has been estimated that no more than 50% of all asbestos fibres in the lung form themselves into asbestos bodies. There is some evidence to suggest that the coating of asbestos fibres renders them at least non-fibrogenic. See, for example, RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 15.

<sup>87</sup>Gordon Atherley, “A Survey of Four Studies on the Health Effects of Asbestos,” in Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos, First Public Meeting, Friday, October 31, 1980*, reported by Elizabeth J. Hiscott (Toronto: Royal Commission on Asbestos, 1980), p. 25; and Zielhuis, *Public Health Risks of Exposure to Asbestos*, p. 81. Asbestos warts are generally found only in asbestos workers.

<sup>88</sup>M. Auribault, “Note sur l’hygiène et la sécurité des ouvriers dans les filatures et tissages d’amiante,” *Bulletin de l’Inspection du Travail* 14 (1906): 126.

<sup>89</sup>H. Montague Murray, “Statement Before the Committee in the Minutes of Evidence,” in *Report of the Departmental Committee on Compensation for Industrial Disease* (London: His Majesty’s Stationery Office, 1907), pp. 127–128.

<sup>90</sup>“Effect of Asbestos Dust on Workers’ Health in Asbestos Mines and Factories,” *Labour Gazette*, Canada (February 1912): 761–762.

on account of the assumed health-injurious conditions of the industry.”<sup>91</sup> We note parenthetically that the practice referred to by Dr. Hoffman has not become a policy of insurance companies.<sup>92</sup>

Dr. W.E. Cooke, in a note in the *British Medical Journal* in 1924, described the death from pulmonary fibrosis of a 33 year old woman who had worked in an asbestos textile factory for 20 years. In a further report on the same case in 1927, Dr. Cooke gave the disease its current name, asbestosis.<sup>93</sup> An investigation of the condition of textile factory workers was undertaken in Great Britain in 1928 and 1929, and the resulting report to Parliament in 1930 recognized “. . . the fact that the inhalation of asbestos dust over a period of years results in the development of a serious type of fibrosis of the lungs.” The report recommended the remedy of dust suppression which led to the promulgation of the Asbestos Industry Regulations in 1931.<sup>94</sup> This was the first regulation of which we are aware that was specifically directed to the control of asbestos in the workplace.

In the United States, a series of clinical reports in the first part of the 1930s confirmed the frequent occurrence of asbestosis among asbestos

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<sup>91</sup>Frederick L. Hoffman, “Mortality from Respiratory Diseases in Dusty Trades (Inorganic Dusts),” *Bulletin of U.S. Bureau of Labor Statistics*, no. 231, Industrial Accidents and Hygiene Series, no. 17 (Washington, D.C.: U.S. Bureau of Labor, June 1918), p. 178.

<sup>92</sup>Dr. Hoffman’s observation received a great deal of attention in submissions to this Commission. See, for example, Ontario Federation of Labour, Written submission to the Royal Commission on Asbestos, #35, January 1981, p. 7; United Electrical, Radio and Machine Workers of America, Written submission to the Royal Commission on Asbestos, #36, January 1981, pp. 2-3; and Canadian Centre for Occupational Health and Safety, Written submission to the Royal Commission on Asbestos, #44, January 1981, p. 5. It may be that this observation had some historical validity. However, we are satisfied that it has not represented the practice of insurance companies conducting business in Canada for a very considerable period of time. Our staff made enquiries of eighteen insurers and in no case did an insurer indicate that an individual was denied coverage because he was an asbestos worker. Generally, insurers indicated they do not impose extra mortality premiums for life insurance solely on the ground of employment in work involving exposure to asbestos. The health history of an individual asbestos worker may, however, result in higher premiums.

<sup>93</sup>W.E. Cooke, “Fibrosis of the Lungs Due to the Inhalation of Asbestos Dust,” *British Medical Journal* 2 (1924): 147; and W.E. Cooke, “Pulmonary Asbestosis,” *British Medical Journal* 2 (3 December 1927): 1024. See also, the discussion in Selikoff and Lee, *Asbestos and Disease*, pp. 22ff.

<sup>94</sup>E.R.A. Merewether and C.W. Price, *Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry* (London: His Majesty’s Stationery Office, 1930); and U.K., Asbestos Industry Regulations (1931), Statutory Rules and Orders, 1931, no. 1140 (London: His Majesty’s Stationery Office, 1931).

workers.<sup>95</sup> These reports were followed by two industry-wide studies. In the first, undertaken by the Metropolitan Life Insurance Company at the request of industry, Lanza, McConnell, and Fehnel reported that two-thirds of the x-ray films of 126 persons selected more or less at random from workers with more than three years' employment in asbestos mines and mills in Quebec and in fabricating plants along the Atlantic coast were not normal.<sup>96</sup> In the second, Dreessen et al. reported in 1938 on findings among 541 workers in asbestos textile plants in North Carolina. Although the study was seriously flawed because it included many newly hired employees and did not fully take account of the workers who had already left the workplace due to disability, there were still numerous demonstrated cases of asbestosis, particularly among those most heavily exposed.<sup>97</sup>

On the compensation side, in the United States, the first formal claim for compensation associated with asbestos exposure was made in 1927. In 1933, Johns-Manville Corporation, even then a leading asbestos producer, settled 11 asbestosis claims out of court for a total sum of \$35,000.<sup>98</sup> Compensation for disease resulting from asbestos became possible in Ontario in 1926, when pneumoconiosis was recognized by *The Workmen's Compensation Act*;<sup>99</sup> and in 1942, the Board allowed its first claim for asbestosis.<sup>100</sup> Thus, it could be said that by the time of World War II, the hazard of asbestos as a pneumoconiotic dust was generally recognized.<sup>101</sup>

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<sup>95</sup>See, for example, K.M. Lynch and W.A. Smith, "Asbestos Bodies in Sputum and Lung," *Journal of the American Medical Association* 95 (30 August 1930): 659-661; R.G. Mills, "Pulmonary Asbestosis — Report of a Case," *Minnesota Medical Journal* 13 (1930): 495-499; J. Donnelley, "Pulmonary Asbestosis," *American Journal of Public Health* 23 (December 1933): 1275-1281; and P. Ellman, "Pulmonary Asbestosis: Its Clinical, Radiological, and Pathological Features, and Associated Risk of Tuberculosis Infection," *Journal of Industrial Hygiene* 15 (July 1933): 165-183.

<sup>96</sup>A.J. Lanza, W.J. McConnell, and J.W. Fehnel, "Effects of the Inhalation of Asbestos Dust on the Lungs of Asbestos Workers," *Public Health Reports* 50:1 (4 January 1935): 1-12.

<sup>97</sup>Waldemar C. Dreessen et al., *A Study of Asbestosis in the Asbestos Textile Industry*, U.S. Public Health Bulletin, no. 241 (Washington, D.C.: U.S. Government Printing Office, August 1938), pp. 68-69. Only 13% of the group surveyed had 10 or more years' employment and only 3 persons had worked more than 20 years. Moreover, it was later reported by Lynch and Ayer of the U.S. Public Health Service that "... the incidence of frank disease was so great that prior to the beginning of the study, out of a total of less than 600 employees, the plants discharged 150 workers suspected of having asbestosis." Only a portion of this group was examined. See Nicholson, "Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States," p. 293.

<sup>98</sup>*Asbestos Litigation Reporter*, 7 February 1979.

<sup>99</sup>Pneumoconiosis was added to Schedule 3 by amendment to Regulation 94, 1 June 1926.

<sup>100</sup>The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 14.

<sup>101</sup>*Borel v. Fibreboard Paper Products Corporation* at 1083, where the court stated in 1973 that "Asbestosis has been recognized as a disease for well over fifty years. . . . By the mid-1930s, the hazard of asbestosis [sic] as a pneumoconiotic dust was universally accepted."



There are two observations we might make concerning the recognition and incidence of asbestosis. First, there was a decades-long delay between the time asbestos was first identified as being able to cause fibrosis and the time fibrosis was generally accepted as a health hazard of asbestos exposure. This delay was no doubt due at least in part to the latency period between first exposure and the clinical manifestation of the disease. Second, the available evidence strongly suggests that the historical incidence of asbestosis was associated with heavy dust exposures. There are many reports of the dusty conditions in asbestos plants in the 1920s and 1930s; ventilation and other dust suppression equipment was minimal or non-existent.<sup>102</sup> In many instances, these dusty conditions persisted right through to the 1960s, and where this occurred the toll of victims of asbestosis remained high.<sup>103</sup>

While the earlier recognition of the disease of asbestosis signalled the dangers of asbestos exposure, the later appraisal of factors causing cancer led to the identification of asbestos as a cause of this disease as well. Significantly, as historic exposure levels in workplaces have been reduced over time, cancer has overtaken asbestosis as the disease of concern resulting from asbestos exposure.

Attention was first called to the possible carcinogenic potential of asbestos in the mid-1930s with a series of medical reports both in the United States and in Great Britain associating asbestos exposure with the

<sup>102</sup>For example, Dr. Irving J. Selikoff and Dr. Douglas H.K. Lee have stated that "Before 1930, conditions in the mines, mills, and factories were largely uncontrolled so that some heavy exposures occurred. Eyewitnesses speak of being unable to see more than a few feet, for instance, because of the amount of dust in the air." See Selikoff and Lee, *Asbestos and Disease*, p. 170. In his testimony before this Commission, Dr. J.C. McDonald described historical conditions in the Quebec mines in these terms: "[Y]ou couldn't see the workers . . . literally couldn't see them. They were in a cloud that you couldn't measure. . . . I'm not speaking of throughout, but I'm speaking particularly of the bagging department, where it was sometimes a habit of even getting into the bag and jumping on it, to pack the stuff in." RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 139.

<sup>103</sup>See Nicholson, "Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States." Even with improvements in dust control, some textile operations remained very dusty. See Jeremiah R. Lynch and Howard E. Ayer, "Measurement of Dust Exposures in the Asbestos Textile Industry," *American Industrial Hygiene Association Journal* 27 (September/October 1966): 431-437; and Hilton C. Lewinsohn et al., "Dust Control in a Conventional Asbestos Textile Factory," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 225-241. Dust levels in Canadian mines and mills remained high into the 1950s. See G.W. Gibbs and R.S.J. du Toit, "Environmental Data in Mining," in *Biological Effects of Asbestos*, eds. P. Bogovski et al., IARC Scientific Publications, no. 8 (Lyon, France: International Agency for Research on Cancer, 1973), pp. 138-144.



development of lung cancer.<sup>104</sup> These scattered case reports were augmented by the *Annual Report of the Chief Inspector of Factories for the Year 1947* in Great Britain. All of the cases known to the Inspectorate to have died from asbestosis since 1924 were reviewed, and more than 13% had lung cancer at a time when, in Great Britain, only 1% would have been expected to have the disease.<sup>105</sup> Then, in 1955, in a seminal epidemiological study, Sir Richard Doll documented that among a group of men employed 20 or more years at Rochdale, an asbestos textile plant in Northern England, the risk of lung cancer was increased tenfold.<sup>106</sup> Even so, Braun and Truan, in 1958, in a study for the Quebec Asbestos Mining Association, concluded there was no risk of cancer associated with asbestos exposure among the Quebec miners.<sup>107</sup> But, in the early 1960s, additional occupational studies provided extensive new evidence. For example, in 1963, Mancuso and Coulter confirmed Doll's findings of increased lung cancer mortality from asbestos exposure in a study of a United States asbestos manufacturing company.<sup>108</sup> In a study published in 1964, Selikoff, Churg, and Hammond found excess death rates from lung cancer among a group of New York - New Jersey insulators in the construction and shipbuilding trades to be 7 times expected rates.<sup>109</sup> By the time Dr. Selikoff and others had reported at the Conference on the Biological Effects of Asbestos organized by the New York Academy of Sciences at the end of 1964, few any longer disputed the association between asbestos exposure and carcinoma of the lung.<sup>110</sup> Subsequent studies, many of which will be considered in this Report, have amply confirmed this association.<sup>111</sup> But, as with asbestosis, so with lung cancer, the path from initial identification to full recognition of the association has taken decades.

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<sup>104</sup>K.M. Lynch and W.A. Smith, "Pulmonary Asbestosis III. Carcinoma of Lung in Asbesto-silicosis," *American Journal of Cancer* 14 (1935): 56-64; S.R. Gloyne, "Two Cases of Squamous Carcinoma of the Lung Occurring in Asbestosis," *Tubercle* 17 (October 1935): 5-10; S.R. Gloyne, "A Case of Oat Cell Carcinoma of the Lung Occurring in Asbestosis," *Tubercle* 18 (December 1936): 100-101; and D.S. Egbert and A.J. Geiger, "Pulmonary Asbestosis and Carcinoma, Report of a Case with Necropsy Findings," *American Review of Tuberculosis* 34 (July 1936): 143-146.

<sup>105</sup>See Selikoff and Lee, *Asbestos and Disease*, pp. 26ff.; Selikoff, "Asbestos-Associated Disease," pp. 569ff.; and E.R.A. Merewether, *Annual Report of the Chief Inspector of Factories for the Year 1947* (London: His Majesty's Stationery Office, 1947).

<sup>106</sup>Richard Doll, "Mortality from Lung Cancer in Asbestos Workers," *British Journal of Industrial Medicine* 12 (1955): 81-86.

<sup>107</sup>D.C. Braun and T.D. Truan, "An Epidemiological Study of Lung Cancer in Asbestos Miners," *Archives of Industrial Health* 17 (1958): 634-653.

<sup>108</sup>Thomas F. Mancuso and Elizabeth J. Coulter, "Methodology in Industrial Health Studies, The Cohort Approach, with Special Reference to an Asbestos Company," *Archives of Environmental Health* 6 (January-June 1963): 210-226.

<sup>109</sup>Selikoff, Churg, and Hammond, "Asbestos Exposure and Neoplasia."

<sup>110</sup>*Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): *passim*.

<sup>111</sup>For a review of many of these studies, see Chapter 5, Section B of this Report.

In the 1940s and 1950s, there were scattered medical reports of the development of the third principal disease caused by asbestos exposure: mesothelioma. For example, 2 cases of mesothelioma were included in the 8 lung cancer cases found in the 4,000 asbestos miners in Quebec surveyed by Cartier in 1952.<sup>112</sup> Still, this tumour was thought to occur exceedingly rarely. Then, in 1960, Wagner, Sleggs, and Marchand reported 33 cases of pleural mesothelioma observed since 1956 in South Africa, of which 13 cases occurred among Cape crocidolite miners and 14 among persons living in the neighbourhood of these mines; and all but one had a proven association with asbestos exposure.<sup>113</sup> By the end of 1961, Dr. J. Christopher Wagner and his colleagues had diagnosed a total of 87 pleural and 2 peritoneal mesothelioma cases in South Africa, more than half of whom had never worked in the asbestos industry but had lived in the vicinity of the crocidolite mines and mills.<sup>114</sup> Since that time, the association between asbestos exposure and mesothelioma has been amply demonstrated in studies and case reports from many different countries.<sup>115</sup>

The studies by Dr. Selikoff and by Dr. Wagner were important not only in bringing to the attention of the public the association between asbestos exposure and cancer, but equally in pointing out that the risk of disease was not confined to asbestos workers in mining and manufacturing.

Dr. Selikoff's investigation of the mortality experience of insulators, which he extended to a North America-wide study, vividly showed for the first time the high incidence of asbestos-related disease outside fixed place industry.<sup>116</sup> It brought into view the fact that asbestos insulation workers and, by extension, demolition workers, building maintenance and repair personnel who might be exposed to asbestos during removal operations, and indeed the entire construction industry were at increased risk of asbestos-related disease. It has been estimated that two-thirds of all asbestos used in the United States between 1890 and 1970 was utilized in the construction industry.<sup>117</sup> Control procedures are inevitably more difficult out-

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<sup>112</sup>P. Cartier, "Discussion," *Archives of Industrial Hygiene and Occupational Medicine* 5 (1952): 262.

<sup>113</sup>J.C. Wagner, C.A. Sleggs, and P. Marchand, "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," *British Journal of Industrial Medicine* 17 (1960): 260-271.

<sup>114</sup>J. Christopher Wagner, "Epidemiology of Diffuse Mesothelial Tumors: Evidence of an Association from Studies in South Africa and the United Kingdom," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 575-578.

<sup>115</sup>See Chapter 5 of this Report. See generally, J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence"; and Selikoff and Lee, *Asbestos and Disease*, pp. 241-306.

<sup>116</sup>Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Cancer Risk of Insulation Workers in the United States," in *Biological Effects of Asbestos*, pp. 209-216; and Selikoff, Hammond, and Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976."

<sup>117</sup>Selikoff, "Asbestos-Associated Disease," p. 585.

side fixed workplaces and have been slow in coming to the construction industry. Furthermore, exposures to asbestos, albeit often of short duration, were commonly intense when they occurred. In these circumstances, it is hardly surprising to find a high incidence of disease outside fixed place industry. Further concern about the incidence of disease outside asbestos mining and manufacturing was raised in 1968 when Harries reported 5 cases of pleural mesothelioma among civilian employees of the Royal Navy Devonport Dockyard in Plymouth, England.<sup>118</sup> By 1973, Harries had observed 55 cases of mesothelioma at Devonport, thus confirming shipyard exposures as an important source of asbestos-related disease.<sup>119</sup> Moreover, only 2 of the mesotheliomas found by Harries occurred in asbestos insulation workers; the remainder were among people in other trades — boiler-makers, welders, etc. — who worked in shipyards together with insulation workers but did not themselves use asbestos often.<sup>120</sup> Their exposures to asbestos were at best intermittent or indirect. Following Harries' studies, similar findings were made of asbestos-related disease among shipyard workers employed during and after World War II in the United States.<sup>121</sup> Again, the time interval until clinical manifestation appears to have precluded an earlier discovery of this important source of disease.

Dr. Wagner's observations of mesotheliomas occurring in the vicinity of the South African Cape crocidolite mines among persons who were not occupationally exposed suggested the potential importance of neighbourhood or environmental exposures in the development of this disease. In 1965, Newhouse and Thompson reported 11 cases of mesothelioma in England whose only known asbestos exposure arose from their living in the same household as asbestos workers, indicating yet another source of this

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<sup>118</sup>P.G. Harries, "Asbestos Hazards in Naval Dockyards," *Annals of Occupational Hygiene* 11:2 (1968): 138.

<sup>119</sup>P.G. Harries, "Experience with Asbestos Disease and Its Control in Great Britain's Naval Dockyards," *Environmental Research* 11:2 (April 1976): 261–267. These 55 cases occurred in the period 1964 to September 1973. There was one peritoneal mesothelioma. All the rest were pleural. Further, 156 men were awarded disability pensions for asbestosis in the period 1964–1973.

<sup>120</sup>*Ibid.*, pp. 261–263. One was an asbestos sprayer and the other a lagger. Harries indicated that "Crocidolite asbestos was extensively applied by a spray process to deckheads and bulkheads for environmental insulation from 1940 to 1960. Records show that asbestos dust concentrations of 177–322 f/cc (fibers 2–10 m long) occurred during the spray process. . . ." (p. 261.) See also, Selikoff, "Asbestos-Associated Disease," p. 584.

<sup>121</sup>Irving J. Selikoff and E. Cuyler Hammond, "Asbestos-Associated Disease in United States Shipyards," *Ca — A Cancer Journal for Clinicians* 28:2 (March/April 1978): 87–89; Irving J. Selikoff, Ruth Lilis, and William J. Nicholson, "Asbestos Disease in United States Shipyards," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 295–311; Phillip L. Polakoff, Barry R. Horn, and Oscar R. Scherer, "Prevalence of Radiographic Abnormalities Among Northern California Shipyard Workers," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 333–339; and Jean Spencer Felton, "Radiographic Search for Asbestos-Related Disease in a Naval Shipyard," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 341–352.



disease.<sup>122</sup> Further cases of mesothelioma both from neighbourhood exposures and from family contacts have subsequently been reported.<sup>123</sup>

## F. The General Health Issues of Current Concern

The epidemiological findings in the 1960s evidencing the association between asbestos and cancer and suggesting that the risk of disease was not confined to asbestos workers served to heighten the public awareness of the health hazards of asbestos and intensify investigation of its ill effects. In the 1970s, there appeared a whole host of new epidemiological studies of defined populations exposed to asbestos in one way or another.<sup>124</sup> These studies certainly confirmed the findings of the 1960s, but they also raised new issues for consideration and discussion. There were, for example, certain studies which appeared to implicate the amphiboles as the more hazardous asbestos fibres, particularly in the production of mesothelioma.<sup>125</sup> Thus, fibre type became an issue, and the controversy was over whether chrysotile was less hazardous than amosite and crocidolite or equally hazardous, a controversy of considerable practical importance since approximately 90% of all asbestos currently mined and used is chrysotile. Certain studies of working populations suggested that some industrial processes might be more hazardous than others. Asbestos mining appeared to carry less of a health risk than asbestos manufacturing; and friction materials operations less than textile operations.<sup>126</sup> These apparent differences among fibre types and among industrial processes spurred efforts to determine whether the differences were real and, if so, what accounted for them. Controversy arose over whether the dimension of the fibre — its length and its thinness — or its chemistry explained these differences, or whether it was simply a matter of different dust levels. While these issues were being debated, fixed workplace exposure levels were being gradually reduced both as a result of improvements in equipment and technology and as a result of more stringent regulation. Occupational exposures to asbestos had to a limited extent been regulated in the past, but in light of the historical incidence of disease, it was abundantly clear that past exposure levels had been far too high. It

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<sup>122</sup>Muriel L. Newhouse and Hilda Thompson, "Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area," *British Journal of Industrial Medicine* 22 (1965): 261–269.

<sup>123</sup>See J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," pp. 439–440; Acheson and Gardner, "The Ill Effects of Asbestos on Health," Table 16, p. 69 (showing 35 collected cases of mesothelioma associated with domestic exposure to asbestos); and Henry A. Anderson et al., "Household Exposure to Asbestos and Risk of Subsequent Disease," in *Dusts and Disease*, p. 155 (noting that domestic exposure cases totalled over 50).

<sup>124</sup>The main studies are reviewed in Chapter 5, Section B of this Report.

<sup>125</sup>See the discussion in Chapter 5, Section C of this Report.

<sup>126</sup>For a review of the major studies by industrial process, see Chapter 5, Section B of this Report.



was far less clear that the current, much lower exposure levels were still excessive. Here again, there was controversy and the latency of the asbestos-related diseases precluded an immediate answer to this controversy by direct observation.

The manifest amount of disease that had occurred in the workplace not unnaturally aroused speculation that asbestos exposures, however minimal, could cause disease in the general population. This speculation was fuelled by the incidence of mesothelioma among those not occupationally exposed and therefore thought to have inhaled only small amounts of asbestos. Moreover, opportunities for asbestos exposure among the general populace appeared to be not inconsequential. Asbestos was utilized in many consumer products; fibres were present in the water systems of most metropolitan centres; and many multi-storey schools and office buildings constructed between the 1950s and the early 1970s contained sprayed-on friable asbestos material. Friable asbestos in schools and other buildings raised another area of debate: if the material was left in place, then the occupants, whether school children or office workers, were potentially exposed; but, if the material was removed, then there were significant asbestos control problems for the maintenance and demolition workers involved. And, as all of these issues — involving both fixed and non-fixed workplaces, both occupational and non-occupational exposures to asbestos — were being debated, this Commission was appointed and went about its tasks.

# **Chapter 3    Asbestos in Ontario: An Overview of Health Effects and Occupational Regulation**

## **A.    The Health Effects of Past Exposures in Ontario**

### **A.1    Introduction**

Individuals may potentially be exposed to asbestos fibres in a number of ways. There are two broad categories of exposure to asbestos: occupational and non-occupational. Occupational exposure may take place in fixed workplaces, as in asbestos mining and manufacturing plants; or it may take place in non-fixed workplaces, as in construction activities or in the maintenance, repair, and demolition of buildings containing asbestos. The exposure of workers may be direct and continuous, as in the case of those employed in the manufacture of asbestos-containing products such as friction materials and textiles; or it may be incidental and intermittent, as in the case of those who, while not directly working with asbestos, may nonetheless be exposed because asbestos is present in the workplace (for example, a maintenance worker in a school with asbestos insulation).

Outside the workplace but closely related to it is the case of family members and others associated with asbestos workers who may be exposed because these workers carry asbestos on their clothing, in automobiles, etc. The general public too may be exposed to asbestos in a variety of ways. Public exposure may arise from the presence of asbestos in schools, other buildings, subways, airports, etc. It may also arise from the use of domestic products containing asbestos and from its presence as a building material in the home. In some circumstances members of the public may also be exposed to asbestos if they live in the neighbourhood of asbestos plants, whether they be mines or manufacturing facilities, or in the vicinity of waste disposal sites where asbestos may have been dumped. Finally, the public has

a general environmental exposure to asbestos arising from the presence of asbestos in drinking water and in the air.

It is one thing to list the manner in which persons may be exposed to asbestos. It is quite another thing to assess how many persons have been exposed to asbestos in Ontario and the amount of such exposure. It can be said that everyone has had some exposure to asbestos in drinking water and the outdoor air. Beyond this, even among those occupationally exposed, there are no reliable data which enable us to estimate with any degree of accuracy either the number of workers exposed to asbestos in the past or the level and duration of such workplace exposure. We do know that there has been some asbestos mining in the past in Ontario, and considerably more asbestos manufacturing. We are also aware that Ontario had a shipyard industry during World War II and that, as in the rest of North America, asbestos was extensively used in the construction industry following the war.<sup>1</sup> However, in shipbuilding and in construction there is great uncertainty about how many employees came into contact with asbestos. In fixed place asbestos manufacturing and in mining, where the number of employees in a plant may be a proxy for the number who were actually exposed to asbestos, there is great uncertainty about the levels of exposure. This uncertainty is due in large part to the infrequency of measurements, to the fact that measurement techniques have changed, and perhaps most telling of all, to the fact that the whole method of measurement changed about fifteen years ago from the counting of dust particles to the counting of asbestos fibres.

How much disease has been caused by asbestos in Ontario? We simply do not know. There are no reliable statistics which permit us to estimate with any confidence the full extent of asbestos-related diseases resulting from past exposures. But such data as do exist chronicle a tragic legacy of disease and death resulting from workplace exposures in this province. Two sources of information in particular give us considerable cause for concern about the health effects of past exposures: the number of claims for compensation for industrial disease resulting from asbestos exposure granted by the Ontario Workers' Compensation Board (WCB); and the particular experience of the workforce employed at the Johns-Manville plant in Scarborough, Ontario.

## **A.2 Compensated Asbestos-Related Diseases**

Allowed claims for occupational disease represent only between 1 and 2% of the over 400,000 claims allowed by the Ontario Workers' Compensation

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<sup>1</sup> Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, p. 15; and The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, pp. 50-52.

tion Board annually.<sup>2</sup> But while occupational disease claims represent only a very small proportion of total claims, they represent a considerably larger proportion of death claims. Fatal cancer provides the most striking statistic: deaths due to cancer have in recent years accounted for over 10% of all the compensated fatality claims for workers' compensation in Ontario.<sup>3</sup>

Occupational cancer, including asbestos-related cancer, has been compensable by the Workers' Compensation Board since 1947.<sup>4</sup> Workers' awareness of the health hazards of asbestos and of their right to disease compensation grew gradually from the time of the first reports of Dr. Irving J. Selikoff's work in the mid-1960s.<sup>5</sup> Statistics provided to us by the Board indicate that as at the end of 1980, 111 asbestos-related cancer claims had been granted compensation: 52 for lung cancer, 48 for mesothelioma, 7 for gastrointestinal cancer, and 4 for laryngeal cancer.<sup>6</sup> A table published in an article, four of whose authors are Board physicians, demonstrates that as of 1978, in comparison with other occupational carcinogens, asbestos was associated with a large proportion of compensated cancer claims.<sup>7</sup> (These data are reproduced in Table 3.1.) Even these statistics do not tell the complete story. For while it is impossible to determine what percentage of Ontario's workers or their survivors, justifiably entitled to compensation for asbestos-related cancers, have in fact applied for benefits, it is reasonably certain that the number of claims identified and allowed by the WCB is considerably less than the total eligible for compensation.<sup>8</sup>

<sup>2</sup>Peter S. Barth, *Workers' Compensation and Asbestos in Ontario*, Royal Commission on Asbestos Study Series, no. 2 (Toronto: Royal Commission on Asbestos, 1982), pp. 6.1-6.2. In 1980, there were 411,476 claims allowed by the WCB, of which 7,611 were industrial disease claims.

<sup>3</sup>Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 6.1-6.2. For example, in 1980 there were 324 fatal claims allowed by the WCB, of which 42 were fatal cancer claims.

<sup>4</sup>Alan C. Chovil et al., "Occupational Cancer: Experience in Ontario," *Canadian Medical Association Journal* 125 (1 December 1981): 1237-1238. See also, *The Workmen's Compensation Act*, S.O. 1947, c. 119, s. 1(1)(h)(ii). Cancer in workers exposed to coal tar became compensable by amendment to the statute in 1932. See *The Workmen's Compensation Act*, 1932, S.O. 1932, c. 21, s. 10.

<sup>5</sup>Irving J. Selikoff, Jacob Churg, and E. Cuyler Hammond, "Asbestos Exposure and Neoplasia," *Journal of the American Medical Association* 188 (1964): 22-26. Idem, "The Occurrence of Asbestosis Among Insulation Workers in the United States," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 139-155.

<sup>6</sup>The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 49.

<sup>7</sup>Chovil et al., "Occupational Cancer: Experience in Ontario," Table I, p. 1238 and text, pp. 1238-1239.

<sup>8</sup>See Chapter 14, Section B of this Report for a full discussion of this issue. See also, Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 6.34ff.; and Paul C. Weiler, *Protecting the Worker from Disability: Challenges for the Eighties*, a report submitted to Russell H. Ramsay, Minister of Labour ([Toronto: Ontario Ministry of Labour], April 1983), p. 24.



Table 3.1  
Type of Cancer and Occupational Exposure in 427 Cases in which Claims for Compensation  
were Accepted by the Workmen's Compensation Board of Ontario, 1947 through 1978

Occupational Exposure	Cancer Type					
	Lung	Nasal Sinuses	Mesothelioma	Gastrointestinal Tract	Larynx	Skin
Nickel refinery processes	132	43			8	
Asbestos	38		32	7	3	
Ionizing radiation, mainly from uranium	54					8
Coal tar and hydrocarbons	35					6
Arsenic	24					10
Aniline dyes						5
Other or unknown						18
						2

**Note:** \*Includes cancer of the bladder, leukemia, and melanosarcoma.

**SOURCE:** Alan C. Chovil et al., "Occupational Cancer: Experience in Ontario," *Canadian Medical Association Journal* 125 (1 December 1981): 1238 (Table I).

The first claim for asbestosis that was allowed by the WCB was awarded in 1942.<sup>9</sup> The data supplied to us by the Board indicate that to the end of 1980 the number of allowed claims for asbestosis was 222.<sup>10</sup> An unspecified number of these 222 claimants may have developed another asbestos-related disease.<sup>11</sup> When the total allowed cancer and asbestosis claims are taken together, it becomes evident that asbestos has been one of the major occupational health hazards in this province.

### A.3 The Experience at the Johns-Manville Plant

Viewed in the context of the number of asbestos-related claims compensated by the WCB, the disease experience at the Johns-Manville Scarborough plant is striking. According to statistics provided to us by the Board, of the 222 allowed claims for asbestosis in Ontario as at the end of 1980, 113 came from this single plant.<sup>12</sup> Of the 108 allowed death claims in the province in which asbestosis or mesothelioma were involved, 51 came from the Scarborough plant.<sup>13</sup> The incidence of asbestosis and mesothelioma in this one plant, amounting as it does to half of all such claims compensated by the Board, has dominated the experience of the province. This dominance is even more striking when one considers that the remainder of the claims are spread widely throughout Ontario industry, with only a handful of other firms having been subject to more than one successful claim, and only two — Raybestos-Manhattan (Canada) Ltd. and Holmes Insulations Ltd. — having yielded more than 5 successful claims.<sup>14</sup> A Johns-Manville operation in New Toronto has yielded exactly 5 claims.<sup>15</sup>

One further comparison vividly demonstrates how disastrous the disease experience at the Scarborough plant has been. In response to our

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<sup>9</sup>The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 1.

<sup>10</sup>*Ibid.*, pp. 9-13.

<sup>11</sup>*Ibid.* The actual data supplied by the Board list "Claims Allowed for Asbestosis, Silico-Asbestosis or Mesothelioma, 1942-1980." The total number of these allowed claims is 258. Of this figure, there are 36 allowed claims for mesothelioma without asbestosis, which by subtraction yields the number 222 for allowed claims for asbestosis. Some persons who were initially compensated for asbestosis may subsequently have developed lung, gastrointestinal, or laryngeal cancer, all of which are now compensable by the Board.

<sup>12</sup>The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, pp. 9-13, 20-21; and Letter from Mr. Andy Emmink, Assistant Secretary, Workers' Compensation Board to the Royal Commission on Asbestos, 29 September 1983.

<sup>13</sup>The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, pp. 9-13, 20-21.

<sup>14</sup>*Ibid.*, pp. 9-13.

<sup>15</sup>Telephone communications between Mr. Andy Emmink, Assistant Secretary, Workers' Compensation Board and Royal Commission on Asbestos Staff, September 1983.

request, the WCB provided us with the data that establish the total number of fatal asbestos-related claims that were awarded to Johns-Manville Scarborough workers as of July 1983.<sup>16</sup> The number of these fatal claims is 68. This small plant, which opened in 1948 and whose total annual employment never exceeded 714 workers,<sup>17</sup> has occasioned more deaths from industrial disease than the entire Ontario mining industry, which annually employs over 30,000 workers, occasions from industrial accidents in an average four-year period.<sup>18</sup>

The Johns-Manville Scarborough plant has been extensively studied by Dr. Murray M. Finkelstein of the Ontario Ministry of Labour. This plant began operations in 1948. In one area of the plant, which came to be known as the Transite pipe section, asbestos-cement pipe was manufactured using a mixture of asbestos containing 4 parts chrysotile to 1 part crocidolite.<sup>19</sup> (A small amount of amosite was apparently used for a few years in the 1970s in an effort to improve the filtration properties of the product.)<sup>20</sup> In another area of the plant, rock wool insulation was manufactured using no asbestos.<sup>21</sup>

In 1955, the flex board shop was opened and from that year until 1970 asbestos-cement board was made in this shop using only chrysotile.<sup>22</sup> Then, beginning in 1960, another area of the plant was opened for the

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<sup>16</sup>Letter from Mr. Alex Joma, Secretary, Workers' Compensation Board to the Royal Commission on Asbestos, 25 July 1983.

<sup>17</sup>Letter from Mr. Thomas S. Patterson, Formerly, Director, Corporate Relations, Johns-Manville Canada Inc. to the Royal Commission on Asbestos, 2 September 1983. Total annual employment in the plant reached 714 in 1975. In that year, of the 714 employees, 296 were employed in the Transite pipe section. Altogether the company estimates that approximately 5,000 workers have been employed at this plant.

<sup>18</sup>The average number of fatal industrial accidents which occur annually in Ontario's mining industry is approximately 16. See Canada/Ontario, *The Report of the Joint Federal-Provincial Inquiry Commission into Safety in Mines and Mining Plants in Ontario: Towards Safe Production* (Burkett Report), Kevin M. Burkett, Chairman, vol. 2: *Statistics and Research Reports* (Toronto: April 1981), p. 15.

<sup>19</sup>Murray M. Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," *British Journal of Industrial Medicine* 40 (1983): 138-144. Dr. Finkelstein, in his testimony before us, indicated that the percentage of crocidolite in the final product was approximately 3%. Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 19. Information was also provided to the Royal Commission on Asbestos by Johns-Manville Canada Inc., 1982.

<sup>20</sup>RCA Transcript, Evidence of Mr. Bruce C. Machin, 28 June 1982, Volume no. 45(A), p. 116; Ontario, Royal Commission on Asbestos, Exhibit II-68 [hereafter RCA Exhibit], in RCA Transcript, Evidence of Mr. Bruce C. Machin, 28 June 1982, Volume no. 45(A), p. 62; Manville, Internal Correspondence from Mr. R. Dennis Stevens, Toronto Plant to Mr. Bruce C. Machin, West Mall, 23 June 1982.

<sup>21</sup>Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 138.

<sup>22</sup>Ibid. See also, RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 19-21.

manufacture of an asbestos insulation material, called Thermobestos, using amosite asbestos. The manufacture of both asbestos-cement pipe and asbestos insulation material was discontinued in 1980.<sup>23</sup>

During the first twelve years of its operation, such dust control as existed in the Transite pipe section of the plant was provided by systems installed at the time of its construction.<sup>24</sup> Further attempts to control dust in this part of the plant led to two major ventilation changes. The first, the installation of a new dry-end dust collector system, involved a 1961 capital expenditure of \$139,000 and, according to Dr. Finkelstein, came into operation in 1962. The second, the installation of a new wet-end dust collector and a new fibre handling collector, involved a 1968 capital expenditure of \$640,542 and, again according to Dr. Finkelstein, came into operation in 1970.<sup>25</sup> A double locker system for the employees in the Transite pipe section and new showers were installed in 1976 at an expenditure of \$170,000.<sup>26</sup> The Transite pipe section was closed in 1980.

The morbidity and mortality experience of the workers at the Johns-Manville Scarborough plant have been the subject of two recently published studies by Dr. Finkelstein,<sup>27</sup> who gave testimony before this Commission at its formal hearings in the summer of 1981.<sup>28</sup> His articles, published in 1982 and 1983, and his subsequent work have enlarged upon his oral evidence.

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<sup>23</sup> Ibid.

<sup>24</sup> RCA Exhibit II-67(a), in RCA Transcript, Evidence of Mr. Bruce C. Machin, 28 June 1982, Volume no. 45(A): Johns-Manville, Internal Correspondence from Mr. K.H. Reeve, Toronto Plant to Mr. J.R. Ariss, CPD HQ, Etobicoke, 24 December 1980.

<sup>25</sup> Ibid. In his publications, Dr. Finkelstein has indicated that these major ventilation changes were actually effected in 1962 and 1970. See Murray M. Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," *American Review of Respiratory Disease* 125 (1982): 497. In the unpublished version of this article, Dr. Finkelstein stated that:

Two major ventilation changes were made in the pipe plant over the years of operation. In 1962, the main duct system in the finishing end of the plant was replaced with new fans, and a baghouse was installed. The ventilation capacity was increased from 45,000 to 60,000 cfm. The old finishing end equipment was serviced for use in the forming end, and ventilation capacity there was subsequently increased from 35,000 to 45,000 cfm. The second major change occurred in 1970 when a negative pressure system of 55,000 cfm capacity was installed in the forming end. [Murray M. Finkelstein, "Asbestosis Among Long-Term Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1981, p. 9. (Mimeographed.)]

See also, RCA Transcript, Evidence of Mr. Bruce C. Machin, 28 June 1982, Volume no. 45(A), pp. 54-57.

<sup>26</sup> RCA Exhibit II-67(a), Johns-Manville, Internal Correspondence from Mr. K.H. Reeve to Mr. J.R. Ariss, p. 6.

<sup>27</sup> Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," pp. 496-501. Idem, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," pp. 138-144.

<sup>28</sup> Dr. Finkelstein testified on 28 July 1981 and again on 26 August 1981. See RCA Transcript, Volume no. 24 and Volume no. 30 respectively.



Dr. Finkelstein first studied only those workers who were hired prior to 1960 and were employed by the company for at least 9 years.<sup>29</sup> His cohort thus constituted 339 men, of whom 252 had at least one year's exposure to asbestos (either in the pipe shop, the cement board shop, or in maintenance work), while the remaining 87 worked in the rock wool insulation operation or were otherwise minimally exposed to asbestos. Dr. Finkelstein traced the mortality experience of these workers to the end of 1980. The employees in rock wool insulation had mortality rates similar to those of the general Ontario population, while the group of asbestos-exposed employees had an overall mortality rate double the general Ontario population. Overall cancer mortality rates among the asbestos workers were 5 times greater than would be expected in the general population, and deaths attributable to lung cancer alone were 8 times more frequent than would be expected in the general population.<sup>30</sup> Of the 58 deaths among the 186 production workers exposed to asbestos in the manufacture of asbestos-cement pipe or asbestos-cement board, over half, 30, were either from lung cancer (20 deaths) or mesothelioma (10 deaths);<sup>31</sup> and in the total workforce studied by Dr. Finkelstein, there were no fewer than 16 mesothelioma deaths, only one of whom had reached retirement age by the time of his death.<sup>32</sup> One of our expert witnesses, Dr. Philip E. Enterline of the University of Pittsburgh, testified before this Commission as to the mortality experience of these workers in the following sobering words: "I've seen those data. They are remarkable data. . . . I don't know of anything quite like that in the literature."<sup>33</sup>

Dr. Finkelstein has attempted to estimate the cumulative exposure of the workers in his cohort on the basis of the available measurement data and communications with company industrial engineers, and having regard to the Transite pipe section's major ventilation changes made in 1962 and 1970.<sup>34</sup> Measurement data from the plant were meagre. There were annual dust counts by government or insurance company hygienists in only five of the years between 1948 and 1961. In 1961, Johns-Manville itself began annual dust measurements with an instrument known as the midget impinger.

<sup>29</sup>Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 139.

<sup>30</sup>*Ibid.*, p. 140.

<sup>31</sup>*Ibid.*, pp. 140-141. These figures are based upon "best evidence." In other words, in addition to the official death certificate codings, the author had further information from clinical, pathological, and necropsy reports.

<sup>32</sup>RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 79-80; and 26 August 1981, Volume no. 30, p. 8. Again, this number is based on "best evidence." In the case of mesothelioma, rather than accepting the cause of death as stated on the death certificate, the author, due to the difficulty of diagnosing mesothelioma, had a diagnosis made either by the Canadian Tumour Reference Centre or by Dr. Alexander C. Ritchie, Professor of Pathology at the University of Toronto.

<sup>33</sup>RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 141.

<sup>34</sup>Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 497; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 52-53, 127-129.

Fibre counts on at least a quarterly basis were made by the company, beginning in 1969, utilizing the membrane filter method.<sup>35</sup> Dr. Finkelstein attempted to reconstruct historical exposure levels on the basis of the company's membrane filter counts taken immediately prior to the 1970 ventilation change. He discussed historical conditions with two industrial engineers from Johns-Manville (one who was present when the plant was being built and worked there until 1969, and the other who worked in the plant in the 1950s and designed the 1970 ventilation changes).<sup>36</sup> On the basis of these discussions and of fragmentary historical dust counts, he assumed that exposures between 1948 and 1954 were twice as high as those immediately prior to the 1970 ventilation change, that exposures between 1955 and 1962 were 30% higher, and that exposures after 1962 were at the levels recorded prior to the 1970 change. These assumed exposure levels, together with the recorded asbestos-related disease, permitted Dr. Finkelstein to correlate asbestos exposure levels with disease.<sup>37</sup> He found that, at an estimated cumulative exposure of 100 fibres per cubic centimetre-years (f/cc-yrs), lung cancer mortality rates were elevated over eightfold among the asbestos-exposed workers.<sup>38</sup>

The exposure estimates made by Dr. Finkelstein have been questioned as being too low, and the author himself has fairly admitted they are fraught with uncertainty.<sup>39</sup> One source of uncertainty involves work practices at the plant. Based on the testimony we heard, work practices which were quite indifferent to the hazardous nature of asbestos were common and likely gave rise to sporadic exposures of a very high level. The current

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<sup>35</sup>Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 497; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 127. See also, Chapter 5, Section B of this Report.

<sup>36</sup>RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 52-53; and RCA Transcript, Evidence of Mr. Edgar G. Stevens, 10 August 1982, Volume no. 54, p. 50.

<sup>37</sup>Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 497; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 52-53. Dr. Finkelstein testified that the factor of 2 increase to estimate exposures for the period 1948-1954 was based on his discussions with company personnel and on the generally held view that dust conditions in the plant were "pretty bad" in that period.

<sup>38</sup>Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 143.

<sup>39</sup>RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 56-59. See also, RCA Transcript, Evidence of Mr. Julian Peto, 30 July 1981, Volume no. 25(B), p. 5, where Mr. Peto suggested that it is possible that Dr. Finkelstein's estimates of fibre counts were underestimates, and that there may have been much higher crocidolite levels than his estimates would suggest. See also, letter from Dr. Hans Weill, Pulmonary Diseases Section, Department of Medicine, Tulane Medical Center, New Orleans, Louisiana to the Royal Commission on Asbestos, 21 July 1982, observing the striking uncertainty of the Finkelstein exposure estimates.

Vice-President of Manville Canada Inc. recalled his experience as an employee at the Scarborough plant in 1963 in the following words:

I can recall one of my initial making the rounds with the chap who was union president for many years, that I found out how to work a dust collector by putting my head inside the hopper bin and having somebody turn on the shaker and bury me in the hopper, with asbestos. That would be in 1963, when I was in maintenance . . . which was thought to be quite humorous at the time.<sup>40</sup>

A former employee, Mr. Nick Carrigan, a member of the Asbestos Victims of Ontario, spoke of historical conditions in these terms:

In the old days, we had a wheelabrator system where it worked on a timer. At certain times the dust system was shut down, at break periods in particular, and we had what we call . . . we put a vibrator . . . we pressed a button, a vibrator went on the bins to shake the bags. You wouldn't believe . . . you couldn't see yourself. You couldn't see the next guy sitting beside you. And while this was going on, we sat down and we ate our lunch in it.

Now we also willowed blue asbestos fibre, mined in South Africa, which is supposed to be the most deadly of them all. We willowed that raw fibre into what we called a spare bin, and then we handled it. We pulled it out of there without masks, without protective clothing, we pulled that blue asbestos fibre out into buckets and dumped it into the back of the machine.

We had leaks, unbelievable asbestos leaks, all over the place, never fixed. That only came in later years when the pressure was put on a little.<sup>41</sup>

Still, the company's own personnel to whom Dr. Finkelstein submitted his exposure estimates thought they were not unreasonable, and Dr. Finkelstein himself suggested that they are at least accurate to within a factor of 3 to 5.<sup>42</sup> It should be noted that the underlying data on which these

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<sup>40</sup>RCA Transcript, Evidence of Mr. Bruce C. Machin, 28 June 1982, Volume no. 45(A), p. 111.

<sup>41</sup>RCA Transcript, Submission by Mr. Nick Carrigan, Asbestos Victims of Ontario, 16 February 1981, Volume no. 1, p. 180.

<sup>42</sup>Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 143; Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," pp. 497-498; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 56-64.



estimates were based are no more uncertain than the data available for most historical studies of asbestos-exposed workers.<sup>43</sup> As much as anything else, the uncertainty of Dr. Finkelstein's exposure estimates illustrates how little we really know about past levels to which asbestos workers were subjected.

Dr. Finkelstein has now extended his mortality study to include all workers hired before 1960 who were employed one year or more — a total of 535 asbestos-exposed and 205 non-exposed employees — and followed them through 1981.<sup>44</sup> Despite the brevity of the employment criterion for entry to the cohort, the disease incidence remains extraordinarily high. The overall cancer mortality rate is nearly 4 times greater than that of the general population, the lung cancer rate alone nearly 5 times greater, and the rate for gastrointestinal cancer nearly 3 times greater.<sup>45</sup> A comparison of the results of Dr. Finkelstein's study with published studies of other asbestos workers such as those at Rochdale, England, and Charleston, South Carolina, forces us, regretfully, to conclude that the disease experience of the workforce at this plant is in line with the worst asbestos-related health experiences observed anywhere in the world.<sup>46</sup> Indeed, it constitutes nothing less than a world-class occupational health disaster.

This world-class occupational health disaster has not yet run its course. There is a distinct possibility, indeed probability, that more asbestos-related deaths are yet to come among the workers who were employed at this plant. Asbestos-caused diseases do not generally manifest themselves until at least 10 to 15 years after initial exposure and more often occur more than 20 years from the time of first exposure to asbestos. The mortality experience of the Johns-Manville workers reported by Dr. Finkelstein is the result of asbestos exposures many years ago. Dust levels were indeed reduced in this plant over time, and this we expect will have some effect in

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<sup>43</sup> See the discussion of the uncertainty of historical exposure data in Chapter 4, Section D of this Report. See also, the review of the major studies in Chapter 5, Section B of this Report.

<sup>44</sup> Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983. (Mimeographed.)

<sup>45</sup> *Ibid.*, Table 2.

<sup>46</sup> See, for example, John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1-4 (1982): 869-887; Julian Peto, "The Hygiene Standard for Chrysotile Asbestos," *The Lancet* 1 (4 March 1978): 484-489; Julian Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 703-711; and Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 829-836. See also, RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 123.



reducing the incidence of disease.<sup>47</sup> But we must recognize the possibility that as time goes on, the toll of deaths from the past inhalation of asbestos will continue to mount. Telling evidence that this has been happening is found in the continuing work of Dr. Finkelstein who, by August 1982, had observed 21 deaths from mesothelioma among the study population at the plant, an increase of 5 in the 12 months that had passed from the time he last testified before the Commission.<sup>48</sup> All but one of the 21 victims were exposed both to chrysotile and crocidolite asbestos in the pipe plant or in maintenance work.<sup>49</sup> At this time, we can only speculate as to what the future holds for the other plant employees, but we cannot fail to observe that at most they are at a distance of but 35 years from first exposure to asbestos at this plant, and that the employees hired in 1960 or later, who have yet to be studied by Dr. Finkelstein, have barely reached, if at all, 20 years from first exposure.

## B. History of Occupational Regulation of Asbestos in Ontario

Occupational exposure to asbestos in Ontario has been regulated by a combination of: (i) legislative provisions generally directed to the health of workers in the mining, construction, and industrial sectors of the economy; (ii) legislative provisions specific to asbestos; and (iii) non-legislative guide-

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<sup>47</sup>Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 497; and Johns-Manville Canada Inc., Written submission to the Royal Commission on Asbestos, #18, 15 January 1981, Appendix A.

<sup>48</sup>Murray M. Finkelstein, "Mortality in Asbestos-Cement Factory Workers," paper presented at the Second International Symposium on Epidemiology in Occupational Health, Montreal, 25 August 1982, p. 4. When Dr. Finkelstein first testified before this Commission in July 1981, he gave evidence that there were in total 15 mesothelioma deaths of which he was aware among the Johns-Manville workers. RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 79-80. When Dr. Finkelstein returned to complete his testimony in August 1981, he had discovered another mesothelioma, bringing the total to 16. RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 26 August 1981, Volume no. 30, pp. 2-8.

<sup>49</sup>Finkelstein, "Mortality in Asbestos-Cement Factory Workers," pp. 4-5. Dr. Finkelstein, in his September 1983 article, noted:

There were 21 deaths from mesothelioma in the cohort; 19 have had pathological review. Seventeen of these deaths occurred among the production workers with 2 each among the maintenance and control groups. One of the control group spent the first part of his employment in Rock Wool/Fibreglass and was transferred into the Pipe shop after the 1961 classification date; the other man is not recorded as having worked in the asbestos areas. It was possible, however, for men to be assigned to the Pipe shop for brief cleanup duties and he may have been exposed in this fashion. (Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," pp. 9-10.)

lines directly applicable to asbestos exposure and use.<sup>50</sup> As we shall discern, from the 1940s onwards regulatory practices in the United States and the United Kingdom had a considerable influence in Ontario.

Heavy concentrations of dust in the workplace were recognized as a health hazard long before asbestos was widely used. For example, *The Ontario Factories Act* of 1884 provided that all ventilation in factories must "... render harmless, so far as is reasonably practicable, all the gases, vapours, dust or other impurities generated. . . ."<sup>51</sup> An amendment to *The Mining Act* in 1912 provided that dust be removed where present in the air in a quantity "injurious to health"<sup>52</sup> and a similar amendment was added to *The Factory, Shop and Office Building Act* in 1913.<sup>53</sup> This general legislative framework in both the industrial and mining sectors remained in place with minor amendments until the 1960s and was available to control asbestos exposures once asbestos was identified as a serious occupational hazard. However, within this legislative framework there were no specific exposure limits for asbestos. Equally important, the legislation which regulated the construction industry where asbestos was widely used, *The Buildings Trades Protection Act*, 1911, focused almost exclusively on safety and made no reference to health hazards whatsoever.<sup>54</sup>

In 1946, the American Conference of Governmental Industrial Hygienists (ACGIH), a professional organization in the United States, voluntarily assumed the task of formulating recommended occupational health standards, a task which was to influence occupational health policy in Ontario for the next quarter of a century. Acting on the recommendations of Dreessen et al. in 1938 following a study of 541 workers in four asbestos textile plants, the ACGIH adopted a workplace exposure limit, for which it registered the trademark name Threshold Limit Value (TLV). The TLV for asbestos was 5 million particles per cubic foot (mppcf) and was a time-weighted average concentration based on impinger sampling and

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<sup>50</sup> For a general history of occupational regulation of asbestos, see Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, pp. 10-25; and Sandra Glasbeek, *A Survey of Asbestos Policies in Canada with Particular Emphasis on Ontario*, Royal Commission on Asbestos Background Paper Series, no. 1 (Toronto: Royal Commission on Asbestos, 1981).

<sup>51</sup> *The Ontario Factories Act*, 1884, 47 Vict., c. 39, s. 11(3).

<sup>52</sup> *The Mining Amendment Act 1912*, 2 Geo. V, c. 8, s. 18, par. 59.

<sup>53</sup> *The Factory, Shop and Office Building Act*, 1913, 3-4 Geo. V, c. 60, s. 43(13).

<sup>54</sup> *The Buildings Trades Protection Act*, 1911, 1 Geo. V, c. 71.

counted by optical microscopy.<sup>55</sup> The asbestos TLV of 5 mppcf remained operative as a guideline in the United States until 1970. In 1947, the Occupational Health Branch of the Ontario Ministry of Health began using this TLV of 5 mppcf as a criterion in issuing control orders and in making recommendations to employers following air sampling.<sup>56</sup> As in the United States, this TLV was not in and of itself a legally enforceable standard. Rather, it served as a guide to the proper control of dust in the workplace.<sup>57</sup> Moreover, it dictated a count of all dust particles present in the workplace even while serving as a guideline for the control of asbestos where present. The TLV of 5 mppcf remained the recommended standard for fixed place industry in this province for 20 years,<sup>58</sup> thus spanning most of the period for which the Johns-Manville workers studied by Dr. Finkelstein were exposed.

Meanwhile, in 1962, *The Construction Safety Act* came into force and gave recognition for the first time to health hazards in the construction industry.<sup>59</sup> A regulation made under that Act provided that no worker was to be present where there was a hazard of injury from inhaling a noxious gas, fume, or dust unless he was protected by mechanical ventilation or the wearing of a suitable respirator.<sup>60</sup> A further regulation under that Act made in 1973 established conditions under which asbestos sprays could be applied which were so stringent that they quickly led to their elimination.<sup>61</sup>

As for fixed place industry, *The Factory, Shop and Office Building Act* was repealed in 1964 and replaced by *The Industrial Safety Act, 1964*.<sup>62</sup> This new statute gave the Minister of Labour far more extensive powers to deal with exposures to toxic substances in industry. A regulation passed under this Act made specific reference to asbestos as one of the air con-

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<sup>55</sup> Waldemar C. Dreessen et al., *A Study of Asbestosis in the Asbestos Textile Industry*, U.S. Public Health Bulletin, no. 241 (Washington, D.C.: U.S. Government Printing Office, August 1938): 68–69. This study has been severely criticized because of its apparent flaws. Only 13% of the group surveyed had been employed 10 or more years and only 3 workers had been employed more than 20 years. Further, prior to the study the plants apparently discharged 150 workers suspected of having asbestosis. See William J. Nicholson, "Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States," *Annals of the New York Academy of Sciences* 329 (26 October 1979): 293–299.

<sup>56</sup> Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, p. 17.

<sup>57</sup> In other words, there was no specific statutory provision which created an offence for exceeding the control limit. However, the government could prosecute an employer for failure to control dust and use as evidence the fact that the Threshold Limit Value had been exceeded. This illustrates the difference between a legally enforceable standard and a guideline.

<sup>58</sup> Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, pp. 17–18.

<sup>59</sup> S.O. 1961–1962, c. 18.

<sup>60</sup> O. Reg. 269/69, s. 31, revised under *The Construction Safety Act, 1961–62*.

<sup>61</sup> O. Reg. 419/73.

<sup>62</sup> S.O. 1964, c. 45.



taminants which must be exhausted from the workplace in such a manner as to prevent its return.<sup>63</sup> The Act and regulations were revised in 1971–1972, broadening the control over fixed workplace exposures to asbestos.<sup>64</sup>

In the late 1960s, as the technology of measurement improved, the method of measuring workplace exposure to asbestos changed from a count of dust particles to a count of fibres. For measurement purposes an asbestos fibre was defined in accordance with common international practice as a fibre longer than 5 microns<sup>65</sup> whose length to diameter ratio (aspect ratio) was at least 3 to 1. This definition originated in the United Kingdom, having been somewhat arbitrarily selected by three asbestos manufacturers who collaborated on the matter. It was apparently chosen to facilitate counting on the standard instrument of measurement, the optical microscope, and because it was thought that the development of asbestosis was related to longer fibres.<sup>66</sup>

In 1968, the ACGIH referred for the first time to fibre counts as well as particle counts and published a new intended TLV for asbestos of 2 mppcf or 12 fibres per cubic centimetre (f/cc). Two years later, in 1970, the ACGIH, now referring to fibre counts alone, proposed a new TLV of 5 f/cc applicable to all types of asbestos and officially adopted this value in 1973.<sup>67</sup> The Ontario Ministry of Health accepted the proposed values pub-

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<sup>63</sup> O. Reg. 196/64, s. 20(c), under *The Industrial Safety Act, 1964*.

<sup>64</sup> *The Industrial Safety Act, 1971*, S.O. 1971, c. 43; and O. Reg. 259/72.

<sup>65</sup> The correct term for one-millionth of a metre is micrometre, but we will use the more familiar term micron.

<sup>66</sup> This definition was also adopted in light of the evidence then available which suggested that the particle size distribution of an asbestos dust cloud was reasonably constant over a wide range of textile processes. Later work indicated this might not be true. See S. Holmes, "Developments in Dust Sampling and Counting Techniques in the Asbestos Industry," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 289; and C.G. Addingley, "Asbestos Dust and Its Measurements," *Annals of Occupational Hygiene* 9 (April 1966): 73–82. The three asbestos manufacturers were Turner Brothers Asbestos Co. Ltd. (Rochdale), British Belting and Asbestos Ltd. (Yorkshire), and Cape Asbestos Co. Ltd. (Barking), who together formed the Asbestosis Research Council to consider problems of dust sampling as well as the medical aspects of asbestosis. See also, RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 47; RCA Transcript, Evidence of Dr. Graham W. Gibbs, 13 November 1981, Volume no. 33, pp. 29–30; Eric J. Chatfield, "Measurement of Asbestos Fibres in the Workplace and in the General Environment," in Mineralogical Association of Canada, Université Laval, Québec: 20–22 May 1979, *Short Course in Mineralogical Techniques of Asbestos Determination*, ed. R.L. Ledoux (Toronto: Mineralogical Association of Canada, Royal Ontario Museum, May 1979), p. 112; Murray M. Finkelstein, "On an Occupational Standard for Exposure to Asbestos," Appendix 9 to Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, Appendices, p. 74.

<sup>67</sup> Irving J. Selkoff and Douglas H.K. Lee, *Asbestos and Disease* (New York: Academic Press, 1978), pp. 443ff.; and Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, pp. 18–19.



lished by the ACGIH in making assessments of asbestos exposures in Ontario industry. The Ministry of Labour, utilizing the findings of the Ministry of Health, continued to issue orders on the basis of these assessments.<sup>68</sup>

But while the ACGIH was reformulating its TLVs in the late 1960s, a study in the United Kingdom was published which was to have a significant effect on the regulation of asbestos in North America. Asbestos had been regulated in the United Kingdom since 1931 by the Asbestos Industry Regulations passed under the "Dangerous and Unhealthy Industries" section of the *Factory and Workshop Act* of 1901.<sup>69</sup> These regulations contained extensive provisions for dust control but not exposure limits. In 1968, the Committee on Hygiene Standards of the British Occupational Hygiene Society (BOHS) recommended that occupational exposures to chrysotile asbestos be limited to 2 f/cc and occupational exposures to crocidolite be reduced to 0.2 f/cc.<sup>70</sup> The BOHS recommendation for chrysotile was predicated on reducing asbestos exposures to the point where the risk of contracting early signs of asbestosis would be only 1% after a lifetime exposure of 50 years. It was based upon a review of available literature and, in particular, upon a BOHS study of employees in the British asbestos textile mill at Rochdale.<sup>71</sup> The BOHS workplace recommendation of 2 f/cc was subsequently subjected to considerable criticism, but nevertheless it soon served as a standard for many other jurisdictions, among them the United States and Ontario.<sup>72</sup> In the United States, the 2 f/cc standard was recommended by the National Institute for Occupational Safety

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<sup>68</sup> Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, pp. 17-19.

<sup>69</sup> U.K., Asbestos Industry Regulations (1931), Statutory Rules and Orders, 1931, no. 1140 (London: His Majesty's Stationery Office, 1931), under the *Factory and Workshop Act*, 1901.

<sup>70</sup> British Occupational Hygiene Society, Committee on Hygiene Standards, "Hygiene Standards for Chrysotile Asbestos Dust," *Annals of Occupational Hygiene* 11 (1968): 47-48.

<sup>71</sup> *Ibid.*, Appendix (i), pp. 54-58.

<sup>72</sup> See particularly, Peto, "The Hygiene Standard for Chrysotile Asbestos"; Geoffrey Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," *British Journal of Industrial Medicine* 36 (May 1979): 98-112; RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 54; RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 49; and RCA Transcript, Evidence of Mr. Julian Peto, 30 July 1981, Volume no. 25(B), pp. 25-28. The British Occupational Hygiene Society's 2 f/cc standard has been criticized, first because it was a standard based on the prevention of asbestosis only and not cancer, and second because it failed to take adequate account of the fact that asbestosis is a progressive disease even in the absence of further exposure. In fact, the population at Rochdale, whose health experience was studied, was an employed population. Accordingly, no account was taken of persons who had been exposed to asbestos but who had either left Rochdale or who had been moved to other areas in the plant prior to the study having been undertaken. Accordingly, persons who had to terminate their exposure to asbestos or their employment altogether because of illness and those persons who developed symptoms of asbestosis after their employment had ceased were not properly considered in the study.

and Health (NIOSH) in 1972 and adopted by the Occupational Safety and Health Administration (OSHA) as the official standard in that country in 1976.<sup>73</sup> Despite subsequent proposals to lower the limit, this remains the standard in effect in the United States, although OSHA promulgated in November of 1983 an Emergency Temporary Standard of 0.5 f/cc, which was subsequently stayed by court order.<sup>74</sup>

Ontario acted on the BOHS recommendation three years earlier than the United States. In 1973, the Ministry of Health abandoned the TLVs of the ACGIH as a reference point for asbestos and adopted as its guideline the time-weighted average exposure limit of 2 f/cc for all types of asbestos.<sup>75</sup> This guideline became effective not only in the industrial sector but in the mining sector as well. Then, in 1975, the Ministry of Health, through its Occupational Health Branch, adopted a guideline of 0.2 f/cc for the amphiboles.<sup>76</sup> For the first time, the Ontario government had adopted a guideline which differentiated exposure limits on the basis of fibre type.

In 1978, following the recommendations of the Royal Commission on the Health and Safety of Workers in Mines, all provincial health and safety legislation in Ontario was consolidated under *The Occupational Health and Safety Act, 1978*, with administrative responsibility vested in the Ministry of Labour.<sup>77</sup> The Act was proclaimed into force on October 1, 1979.<sup>78</sup>

<sup>73</sup> U.S., National Institute for Occupational Safety and Health, *Revised Recommended Asbestos Standard*, prepared by Richard A. Lemen and John M. Dement (Washington, D.C.: U.S. Department of Health, Education and Welfare, December 1976). For a history of U.S. regulatory action, see Nicholson, "Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States," pp. 294-297.

<sup>74</sup> RCA Exhibit II-3, in RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8: Philip E. Enterline, "Epidemiologic Basis for the Asbestos Standard," paper presented at the Second Annual Symposium on Environmental Epidemiology, Pittsburgh, Pennsylvania, 28 April 1981. (Mimeographed.) See also, U.S., Department of Labor, Occupational Safety and Health Administration, "Occupational Exposure to Asbestos; Emergency Temporary Standard," 29 CFR Part 1910, 48 FR 51086-51140, 4 November 1983.

<sup>75</sup> Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, p. 19.

<sup>76</sup> Ibid.

<sup>77</sup> S.O. 1978, c. 83.

<sup>78</sup> S.O. 1979, Table of Proclamations, p. 858. Three regulations were passed pursuant to this statute: the Regulation for Industrial Establishments, O. Reg. 658/79 as amended; the Regulation for Construction Projects, O. Reg. 659/79 as amended; and the Regulation for Mines and Mining Plants, O. Reg. 660/79 as amended. (See now, R.R.O. 1980, Regulations 692, 691, and 694 respectively.) Only the Regulation for Mines and Mining Plants contained an enforceable control limit for exposure to asbestos, incorporating by reference for underground mines only the 1979 TLVs of the ACGIH. For asbestos, the TLVs prescribed exposure standards of 0.2 f/cc for crocidolite, 0.5 f/cc for amosite and tremolite, and 2.0 f/cc for chrysotile and all other forms of asbestos.

However, the Ontario government had publicly declared its intention to regulate general occupational health hazards and the exposure of employees to asbestos in particular, under legally enforceable control limits, even before the new legislation was proclaimed.<sup>79</sup> Once in force, section 41 of *The Occupational Health and Safety Act* gave the Lieutenant Governor in Council power to make regulations designating any substance and, once designated, prohibiting, regulating, or limiting the handling, exposure, use, and disposal of such a substance. On June 28, 1980, shortly after the appointment of this Commission, the Ministry of Labour published in *The Ontario Gazette* notice of intent to designate asbestos, together with five other substances and noise.<sup>80</sup> On August 16, 1980, a proposed asbestos regulation was published, together with a request for public comment from workers, employers, and other interested parties.<sup>81</sup> In setting exposure limits under the proposed regulation, the Ontario government again drew upon British advice. In 1979, the United Kingdom Advisory Committee on Asbestos had tabled its final report.<sup>82</sup> Among its recommendations were that the time-weighted average control limits for exposure to crocidolite, amosite, and chrysotile asbestos be 0.2, 0.5, and 1.0 f/cc (over a 4-hour sampling period) respectively.<sup>83</sup> This reflected the view of the Advisory Committee that different fibre types were of different relative pathogenicity; and that crocidolite was the most pathogenic, chrysotile the least, with amosite occupying an intermediate position. The asbestos regulation proposed by the Ontario Ministry of Labour in August of 1980 adopted this approach and these numbers (but over a 40-hour averaging period) and also provided that the maximum allowable concentration in any period of time be 2.0 f/cc for crocidolite and amosite and 5.0 f/cc for chrysotile and all other types of fibres.<sup>84</sup> The proposed regulation on asbestos was revised and ultimately was filed and became effective in law in

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<sup>79</sup> Ontario, Ministry of Labour, "Notice of Intent to Regulate Lead, Asbestos and Silica and Occupational Health Hazards," *The Ontario Gazette*, vol. 111-29, 22 July 1978, p. 3546.

<sup>80</sup> Ontario, Ministry of Labour, "Notice of Intention," *The Ontario Gazette*, vol. 113-26, 28 June 1980, p. 2641.

<sup>81</sup> Ontario, Ministry of Labour, "Proposed Regulation under *The Occupational Health and Safety Act*, 1978: Asbestos — Designated Substance," *The Ontario Gazette*, vol. 113-33, 16 August 1980, pp. 3339-3348.

<sup>82</sup> U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979).

<sup>83</sup> *Ibid.*, Recommendation 14, p. 74, regarding crocidolite; Recommendation 17, p. 78, regarding amosite; and Recommendation 15, p. 77, regarding chrysotile. In fact, the Advisory Committee recommended that there should be a statutory ban on the import of raw crocidolite fibre and that the control limit for crocidolite already in place be 0.2 f/cc.

<sup>84</sup> Ontario, Ministry of Labour, "Proposed Regulation under *The Occupational Health and Safety Act*, 1978: Asbestos — Designated Substance," *The Ontario Gazette*, vol. 113-33, 16 August 1980, s. 4(2) and Schedule, pp. 3340, 3348.



August 1982, more than two years after the notice of intent was published.<sup>85</sup> Under the Regulation, asbestos was prescribed as a designated substance; instead of being simply a guide to proper dust control, the numerical exposure limits became legally enforceable standards; these exposure limits were unchanged from the August 1980 proposal, save for the maximum allowable concentration for crocidolite, which was reduced from 2.0 f/cc to 1.0 f/cc, and for amosite, which was raised from 2.0 f/cc to 2.5 f/cc. And significantly, the employer was placed under an explicit obligation not merely to ensure that the exposure limits were not exceeded but also to ensure that the time-weighted average exposure of workers was reduced to the lowest practical level.<sup>86</sup> The Regulation Respecting Asbestos was made applicable to the industrial and mining sectors and became, at the time of its promulgation, one of the most stringent of any jurisdiction in the world, Sweden being a notable exception.<sup>87</sup>

Asbestos on construction projects is to be subject to a separate regulation. In October 1981, the Ministry of Labour circulated guidelines for the removal and treatment of asbestos on construction projects and published, in August 1982, a Proposed Regulation Respecting Asbestos on Construction Projects,<sup>88</sup> followed by a revised proposal in January 1983.<sup>89</sup> The proposed regulation reflects the difficulties inherent in monitoring exposures to hazardous substances in non-fixed site workplaces. Rather than prescribing exposure limits for the control of asbestos, it sets out a detailed list of work

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<sup>85</sup> Regulation Respecting Asbestos, O. Reg. 570/82, made under the *Occupational Health and Safety Act*, R.S.O. 1980, c. 321. The mining regulation has not been changed so that in theory the TLVs of the ACGIH still apply to underground mines. But we are advised that it is the intent of the Ontario government to make the Regulation Respecting Asbestos applicable to all mining and to amend ss. 243 and 244 of the Regulation for Mines and Mining Plants so that s. 279 will be inapplicable in the case of a designated substance.

<sup>86</sup> Regulation Respecting Asbestos, ss. 2 and 4.

<sup>87</sup> Arne Westlin, "The Swedish Experience in Banning and Restricting Certain Applications of Asbestos and in the Use of Substitutes," paper presented at *Engineering Occupational Health*, a symposium sponsored by the Department of Chemical Engineering and Applied Chemistry, University of Toronto, Canadian Society for Chemical Engineering (Toronto Section), Chemical Institute of Canada (Toronto Section), and Ontario Ministry of Labour, Toronto, 13 April 1978. (Mimeographed.) In a copy of a telex to the British Embassy in Washington and dated 24 August 1983, sent by Mr. Cyril D. Burgess of the U.K. Health and Safety Executive to the Royal Commission on Asbestos, it was stated, *inter alia*, that the Health and Safety Commission had decided that: "1. Exposure limits for chrysotile to be reduced to 0.5 fibres per ml and for amosite to 0.2 fibres per ml effective from 1 August 1984. 2. A ban should be placed on the importation and use in manufacture of amosite and products containing it. Prohibition regulations should be brought forward for amosite and crocidolite to come into effect on 1 June 1984. . . ."

<sup>88</sup> Ontario, Ministry of Labour, Occupational Health and Safety Division, "Notice of Proposed Regulation: Designated Substance — Asbestos on Construction Projects," *The Ontario Gazette*, vol. 115-33, Saturday, 14 August 1982, pp. 3194-3197.

<sup>89</sup> Ontario, Ministry of Labour, Occupational Health and Safety Division, "Proposed Regulation Respecting Asbestos on Construction Projects and Related Codes," presented at a Public Meeting, 17 January 1983, Toronto. (Mimeographed.)



practices to be followed — in other words, “regulation by procedure.” As of the summer of 1983, the Proposed Regulation Respecting Asbestos on Construction Projects remained under consideration within the Ministry of Labour.

From this brief history, it can be seen that the Ontario government, in evolving its regulation of occupational exposure to asbestos, has borrowed freely and frequently from practices in the United States and in the United Kingdom. But the history of Ontario’s experience is not only one of sharing U.S. and U.K. practices. It is one that reflects the slow recognition in all countries of workplace environmental factors as determinants of health.

## Chapter 4 Sources of Information on the Health Effects of Asbestos

### A. Introduction

In his address called "A Review of Diagnostic Tests for Causation" at the Second Public Meeting of this Commission held on December 12, 1980, Dr. David L. Sackett, Professor of Clinical Epidemiology and Biostatistics and Professor of Medicine at McMaster University, pointed out that two different kinds of expertise are required in order to understand the health effects of asbestos exposure.<sup>1</sup> First, there is a need for methodological expertise in the critical assessment of information concerning the etiology, that is, the causation of asbestos-related diseases. Second, there is a need for biological expertise in the subject matter of asbestos and its effects on the cells, tissues, and organs of the body.

During the course of our inquiry we heard sworn testimony from a substantial number of the world's acknowledged experts on the health effects of asbestos. These witnesses imparted to us their expertise on the two subjects to which Dr. Sackett referred in his public address. In addition, we have digested a good deal of the voluminous and proliferating medical literature on asbestos. In the next two chapters we report on what we have learned.

In this chapter we first consider the biological evidence, what we know and what we do not know, about the biological mechanisms by which asbestos causes disease. We next discuss in general terms the principal sources of information on the health effects of asbestos and the criteria by

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<sup>1</sup>David L. Sackett, "A Review of Diagnostic Tests for Causation," in Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos, Second Public Meeting, Friday, December 12, 1980*, reported by Lydia Dotto (Toronto: Royal Commission on Asbestos, 1981), p. 10 and Appendix B, p. 1.

which that information ought to be assessed. Then we offer our judgement as to the strengths, weaknesses, and limitations of the available evidence. Our purpose is to provide a foundation which will enable us to address, both in Chapter 5 and in the remainder of this Report, the health issues that are of current and future concern.

## B. The Biological Evidence

We turn first to the biological evidence pertaining to the pathogenesis of asbestos-related diseases. We do so with considerable trepidation. Current understanding of the biological mechanisms which induce asbestos-related cancer or indeed asbestosis is limited. Much remains in the realm of speculation and hypothesis. Our justification for raising the subject is the prospect that what we do know about these mechanisms will assist us to determine the biologically relevant characteristics of asbestos fibres and thereby permit us to formulate recommendations that take account of these characteristics.

### B.1 The Inhalation of Asbestos Fibres

The importance of fibres in terms of their health effects following inhalation depends principally upon the following factors: (i) penetration — the ability of fibres to enter the respiratory system; (ii) deposition — where fibres are deposited within the respiratory system; (iii) retention — the extent to which fibres may be retained at the site of deposition; (iv) removal — the rate of clearance of fibres from the respiratory system; (v) translocation — the movement of fibres to other sites within the body; (vi) possibly durability — the ability of fibres to survive for some period of time rather than being dissolved or breaking up in tissue; and (vii) the biological effects of the fibres.<sup>2</sup>

Whether or not inhaled asbestos fibres will enter the lung depends upon the aerodynamic behaviour of the particles, the dimensions of the

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<sup>2</sup>Graham W. Gibbs and Chung-Yung Hwang, "Physical Parameters of Airborne Asbestos Fibres in Various Work Environments — Preliminary Findings," *American Industrial Hygiene Association Journal* 36:6 (June 1975): 459-460; Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 96; Victor Timbrell, "The Inhalation of Fibrous Dusts," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 255-273; and Victor Timbrell, "The Inhalation of Fibres," in *Pneumoconiosis: Proceedings of the International Conference, Johannesburg, 1969*, ed. H.A. Shapiro (Cape Town: Oxford University Press, 1970), pp. 3-9.

respiratory tract they enter, and the breathing patterns of the individual.<sup>3</sup> Dr. Victor Timbrell and his colleagues at the Medical Research Council Pneumoconiosis Unit at Penarth, Wales, have demonstrated that the aerodynamic behaviour of particles or their settling rate is a function mainly of the diameter of the fibre.<sup>4</sup> Dr. Timbrell has shown that the maximum diameter of respirable particles is between 3 and 3.5 microns.<sup>5</sup>

Once fibres enter the respiratory system, three factors come into play:

- (i) Diameter — long fibres, if thin enough, can avoid being deposited by gravitational settlement or inertial precipitation in the nasal passages or high in the respiratory tract and may travel deeply into the lung.<sup>6</sup>
- (ii) Length — the length of the fibre also influences its chance of being intercepted high in the tracheal-bronchial tree before it can be deposited in the lung. (This is because the airway passages along which it must travel are fairly narrow. For this reason, very long fibres, that is, fibres over 100 microns in length, are unlikely to be distributed into the more peripheral or terminal air spaces of the lung.<sup>7</sup> Examination of pathological specimens by electron microscopy indicates that there are virtually no fibres longer than 200 microns in length in the lung and most are less than 50 microns in

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<sup>3</sup>Margaret R. Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," *American Review of Respiratory Disease* 114:1 (July 1976): 193.

<sup>4</sup>Timbrell, "The Inhalation of Fibrous Dusts," p. 273. Dr. Timbrell demonstrated that the important parameter for the main deposition mechanisms is particle free-falling speed, and for a fibre this is predominantly determined by the diameter and not the length of the fibre. This in turn explains the presence of asbestos fibres 50 microns and even 200 microns in length in the lung. See also, RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 97.

<sup>5</sup>*Ibid.* Fibres greater than 3 to 3.5 microns in diameter will not penetrate into the alveolar region of the lung.

<sup>6</sup>Timbrell, "The Inhalation of Fibres," p. 3. Dr. Timbrell explained "... that the free-falling speed of a fibre is approximately proportional to the diameter squared and almost independent of the length. Only if a fibre is of small diameter can it avoid deposition from gravitational settlement and inertial impaction high in the respiratory tract and succeed in penetrating to the pulmonary air spaces."

<sup>7</sup>Timbrell, "The Inhalation of Fibres," p. 3; and RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 96. As Dr. Gibbs explained, as fibres try to pass through airways which are fairly narrow, a fibre which is fairly long and gets out of orientation so that it is angled to the wall will be trapped. Also, if there is a change in direction of airflow, as there would be at a branch, the momentum of the fibre may carry the fibre onto the branch.



length.)<sup>8</sup> (iii) Shape — to the extent that chrysotile fibres are curly in shape, they correspondingly have a greater cross-sectional area and therefore interception at airway branches and in nasal passages is more likely. A curly chrysotile fibre would tend not to get down as deeply into the lung as a straighter, more needle-like amphibole fibre.<sup>9</sup>

For those asbestos fibres that are deposited within the lung, the body has a fairly efficient defence system to deal with them. Two main biological mechanisms participate in the clearance of the fibres from the lower respiratory tract: (i) Most of the dust containing asbestos fibres is removed by the mucociliary escalator of the tracheal-bronchial tree. The material is either coughed up or swallowed. In general, it would appear that short fibres are cleared more readily by this method than long fibres.<sup>10</sup> (ii) Certain cells have the responsibility for defence of the host against all alien materials and these cells produce their effect by their ability to engulf foreign material. These phagocytic, that is, scavenger cells are known as macrophages. Depending upon what the foreign material is, the macrophage, by liberating enzymes within itself, can digest or destroy the

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<sup>8</sup>Paul Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, Gaithersburg, Maryland: 18–20 July 1977, NBS Special Publication 506 (Washington, D.C.: U.S. National Bureau of Standards, November 1978), p. 136; Timbrell, "The Inhalation of Fibrous Dusts," pp. 269ff.; and Marc Trudeau, "Methods for the Evaluation of Asbestos Dust Concentration at the Workplace," in Mineralogical Association of Canada, Université Laval, Québec: 20–22 May 1979, *Short Course in Mineralogical Techniques of Asbestos Determination*, ed. R.L. Ledoux (Toronto: Mineralogical Association of Canada, Royal Ontario Museum, May 1979), p. 215.

<sup>9</sup>See Timbrell, "The Inhalation of Fibres," p. 6, where the author stated that "Curvature in a fibre therefore works to decrease the efficiency of penetration, especially in narrow airways. Looked at in another way, introduction of curvature into a fibre causes deposition to be earlier and the site to be higher in the respiratory tract. The fibres most affected are the long fibres, which are also in general the most massive." See also, Gibbs and Hwang, "Physical Parameters of Airborne Asbestos Fibres in Various Work Environments — Preliminary Findings," p. 459; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 45. Dr. Gibbs is of the view that the curliness of the chrysotile fibre has been somewhat overemphasized. His examination of the airborne fibres that exist in the mines and mills indicated that there was some curliness to the chrysotile fibre but not nearly as much as in the UICC (Union Internationale Contre le Cancer) samples used by Dr. Timbrell. RCA Transcript, Evidence of Dr. Graham W. Gibbs, 13 November 1981, Volume no. 33, pp. 20–21.

<sup>10</sup>RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 19–22; John E. Craighead and Brooke T. Mossman, "The Pathogenesis of Asbestos-Associated Diseases," *The New England Journal of Medicine* 306:24 (17 June 1982): 1448; and Telephone communication between Dr. Jerrold Abraham, Department of Pathology, College of Medicine, State University of New York, Syracuse, New York and Royal Commission on Asbestos Staff, 18 August 1983, together with confirming letter from Royal Commission on Asbestos Staff to Dr. Abraham of same date. Dr. Abraham and Dr. Cecilia Smith, University of California at San Diego, conducted an animal experiment in which rats were exposed to an aerosol of chrysotile taken from the Jeffrey Mine in Quebec. An analysis of the fibres recovered from the lungs of these rats at subsequent time intervals indicated that the short fibres tended to clear the lungs while the long fibres remained.

material. In instances where the macrophage is incapable of destroying the material, as is the case with asbestos, the macrophage can keep the foreign substance from doing any harm by simply engulfing it.<sup>11</sup> It is also thought that the coating of asbestos fibres (asbestos bodies) may occur when the fibres have been engulfed by the macrophages.<sup>12</sup>

Shorter fibres, especially those less than 5 microns in length, are readily and completely engulfed by macrophages, but longer fibres are not; that is, those fibres longer than 5 microns or perhaps 8 microns in length are not completely engulfed by macrophages.<sup>13</sup> The importance of this in terms of disease production will be discussed below.

Macrophages are particularly mobile, and once these scavenger cells pick up asbestos fibres they follow one of three pathways. One pathway is to escape to the mucociliary apparatus, eventually to be coughed up and removed from the lung. A second pathway appears to be into the lymphatic system from which the cells are also removed. A third pathway is to penetrate deep into the tissues of the lung, and this appears to be the dangerous pathway from the point of view of disease production. The evidence is not clear as to why the macrophage chooses one pathway over the other.<sup>14</sup>

It has been estimated that the body's clearance mechanisms are 95 to 98% effective in removing asbestos fibres that have been inhaled into the lung.<sup>15</sup> There is no reliable evidence to indicate whether this clearance rate will vary in accordance with the manner of asbestos exposure. But the possibility does remain that high doses over short periods of time (peak

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<sup>11</sup>RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 19-22; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 7-12.

<sup>12</sup>RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 16-17; and Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," p. 138.

<sup>13</sup>Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 194; RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 24; and A. Morgan, "Effect of Length on the Clearance of Fibres from the Lung and on Body Formation," in *Biological Effects of Mineral Fibres*, vol. 1, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), p. 330.

<sup>14</sup>RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 20-22; and RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 27.

<sup>15</sup>Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 194; and RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 38-39. It has been suggested that there are two phases of clearance through the tracheal-bronchial tree: about half the asbestos is removed within a few days; subsequently, clearance continues for extended periods. The bulk of this material is excreted in the feces. See Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," pp. 1448-1449.

exposures) or indeed continuous, higher levels of exposure will overwhelm the lung's clearance mechanism.<sup>16</sup> A variety of external influences such as cigarette smoke and air pollutants no doubt also affect the lung's clearance mechanisms, but these factors are difficult to assess, in part because individuals appear to differ in their response to inhaled particles. It is also difficult to assess the durability of fibres within the lung. It has been suggested by some authorities that either the ability of chrysotile to separate into tiny fibrils or its tendency to dissolve in weakly acidic environments might make it easier to be cleared from the lungs than the amphibole fibres.<sup>17</sup> Whatever the factor or factors of importance, there does appear to

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<sup>16</sup>RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 46–47. Dr. Becklake thought it likely both on the basis of scattered evidence and on the basis of what is known about lung clearance mechanisms that large, intense doses might permit greater temporary retention of asbestos fibres. See also, RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 41–42; RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, p. 54; and RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 36. Dr. Kotin was of the view that there is a difference biologically in the way one gets a dose of asbestos. He testified that it matters whether one gets a certain amount of dose in a very short period of time, as opposed to the same dose over a longer period, due to the possibility of overwhelming defence mechanisms in the body. See also, RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 61–62. Dr. Weill indicated it is possible that peak exposures will produce more disease because there may not be enough macrophage cells to clear the lung. Dr. John M.G. Davis et al. examined the effects of intermittent, high asbestos exposure (peak dose levels) on the lungs of rats. Their findings appear to refute the idea that very high exposures to asbestos for short periods result in a much higher lung burden of retained dust than might be expected. However, Dr. Davis in his testimony observed that in the experimental situation it was impossible to examine the effects of the realistic differential that one sees in factory situations. In his animal experiments he had only a fivefold difference between “even” and “peak” doses, whereas a peak dose caused by a temporary machine defect in a factory could easily be 100 times higher than the ordinary level. See John M.G. Davis et al., “The Effects of Intermittent High Asbestos Exposure (Peak Dose Levels) on the Lungs of Rats,” *British Journal of Experimental Pathology* 61 (1980): 272–280; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 103–104.

<sup>17</sup>Dr. Alison D. McDonald suggested in her evidence that the amphiboles tend to remain as longer fibres once they get into tissue, whereas chrysotile does tend to split transversally into much shorter pieces. RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 47–48. Dr. Gibbs expressed the view that durability, that is, the ability of the fibre to survive for some period of time prior to either dissolving or breaking up into the tissue, is important. He noted that chrysotile, unlike the amphiboles, is not very acid resistant and that the ability to break apart is far greater for chrysotile than it is for the amphiboles. He suggested these differences might improve the ability of chrysotile to be cleared from the lung more efficiently than the amphiboles. RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 126–132. Dr. Eric J. Chatfield indicated that a fibre of an amphibole which goes into the lung is going to remain there as an amphibole fibre. However, the chemistry of a chrysotile fibre gives it the potential to be dissolved in the lung. RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 66–67. See also, RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 55–56.



be evidence to indicate a preferential clearance of chrysotile as opposed to amphibole fibres from body tissue.<sup>18</sup>

## B.2 The Ingestion of Asbestos Fibres

While inhalation is the principal method by which asbestos fibres enter the body, they may also enter through ingestion, either in food, beverages, drinking water, or drugs. In contrast to inhalation, there is no limitation on either the length or diameter of an asbestos fibre that is capable of being ingested. In theory, fibres of any size or shape might be present in food or beverages or drinking water and therefore capable of being swallowed; in practice, the sizes of fibres that are ingested are typically much smaller than those that are inhaled, averaging around 2 microns in length.<sup>19</sup>

It would appear that fibres entering the body through inhalation and ingestion are dealt with quite differently. Fibres which are swallowed tend to pass right through the gastrointestinal tract and be immediately removed. Fibre penetration of the tissue or lining of the gut following ingestion is speculative at best.<sup>20</sup> On the other hand, fibres which are inhaled and are too long to be engulfed by macrophages are not removed but remain in the body and accordingly have ample opportunity to penetrate the tissue of various organs.

## B.3 The Pathogenesis of Asbestosis

Fibrosis is the formation of fibrous or scar tissue, usually as a reparative or reactive process. It was originally thought that the fibrogenic effect of asbestos fibres was due either to the cells in the lungs being physically irritated by the fibres or to the insolubility of fibres in tissue.<sup>21</sup>

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<sup>18</sup>Margaret R. Becklake, "Exposure to Asbestos and Human Disease," *The New England Journal of Medicine* 306:24 (17 June 1982): 1481.

<sup>19</sup>See Chapter 11 of this Report. See also, RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 97.

<sup>20</sup>See Chapter 5, Section I of this Report. See also, R.E. Bolton and J.M.G. Davis, "The Short-Term Effects of Chronic Asbestos Ingestion in Rats," *Annals of Occupational Hygiene* 19:2 (November 1976): 121-128; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 97-100. Dr. Davis testified that he has examined the gastrointestinal tissue and other tissue of animals that had ingested asbestos for their full lifespan and could not find any asbestos, let alone any gastrointestinal or other tumours. See also, J.M.G. Davis, R.E. Bolton, and J. Garrett, "Penetration of Cells by Asbestos Fibers," *Environmental Health Perspectives* 9 (1974): 255-260; and World Health Organization, International Agency for Research on Cancer Working Group, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, Lyon, France: 14-17 December 1976 (Lyon: IARC, 1977), pp. 59-60.

<sup>21</sup>Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," p. 139.



Recent studies have led to other hypotheses based on the response of the macrophage. Asbestos fibres totally engulfed within the macrophage are relatively immobile and not likely to be harmful. If, however, the fibre is liberated, then either the fibre or the process by which it is liberated may produce an adverse health effect. Fibres attacked by macrophages may be liberated in one of two ways. First, a totally engulfed fibre will survive within the macrophage and the macrophage will try to destroy the fibre by liberating within itself certain enzymes. As contrasted with inorganic materials, these enzymes are not successful in destroying the fibre. Macrophages have a life of 30 to 40 days, and when the macrophage dies the engulfed fibre is freed into the surrounding medium.<sup>22</sup> It may subsequently be re-engulfed by other macrophages. A second way in which the fibre becomes liberated arises if it has a length greater than that of the macrophage itself. Then, the macrophage will be physically incapable of ingesting the entire fibre and the fibre will only be partly engulfed by the macrophage. The macrophage defence system does try to respond to these longer fibres. In circumstances where macrophages cannot completely engulf fibres, the secretion or release of enzymes or other substances may be greater than when the macrophage can completely engulf the fibre. With a long fibre, many macrophages may accumulate and may fuse to form a giant cell.<sup>23</sup>

Beyond this point the pathogenesis of asbestosis is not at all well understood, but one theory has it that the enzymes which leak from incompletely engulfed cells dissolve the protein in the lining of the alveolar sacs, leading to tissue death and fibrosis.<sup>24</sup> Another hypothesis is that the enzymes and other substances released by the macrophages stimulate the proliferation of another type of cell called a fibroblast. In turn, fibroblasts contribute to fibrosis by synthesizing increased amounts of connective tissue such as collagen.<sup>25</sup> One further hypothesis is that some of these fibroblasts actually engulf asbestos fibres in their own right. It is quite possible that asbestos stimulates the production of collagen by various cells within the body, such as fibroblasts, thereby causing fibrosis.<sup>26</sup>

Another way in which the body reacts to asbestos fibres, the importance of which is not entirely clear, is by the formation of asbestos bodies.

<sup>22</sup>RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 25-31.

<sup>23</sup>Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1449; RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 7-12; and RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 25-31.

<sup>24</sup>RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 28-31.

<sup>25</sup>Ibid. See also, Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1449; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 12-15.

<sup>26</sup>RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 12-15, 25.

These bodies are believed to be formed by macrophages that have engulfed fibres.<sup>27</sup> Only a small percentage of all fibres inhaled into the lung do become coated, and it is not known why most of the fibres are not coated. The presence of asbestos bodies does not appear to stimulate fibrosis, and in fact there is evidence that tends to suggest that the coating of fibres renders them at least non-fibrogenic.<sup>28</sup> However, approximately three-quarters of all asbestos fibres remain uncoated. These fibres tend to accumulate in the peripheral regions of the lower lobes of the lungs, the site of early fibrosis.<sup>29</sup> Over time, the fibrotic process progresses in an apparently diffusive manner, affecting more and more of the lung. As the fibrosis becomes extensive, the lung shrinks to below normal size and in cases of advanced asbestosis may take on a "honeycomb" appearance.<sup>30</sup> Figure 4.1 shows a typical lung with fibrosis.

## B.4 Carcinogenesis

What are the mechanisms of asbestos-induced carcinogenesis in humans? The short answer to this question is that the scientific and medical community appears to know very little about these mechanisms. It is thought that asbestos-induced cancer, like other types of cancer, originates from cells which have been transformed, frequently by changes in or damage to the DNA or other genetic material.<sup>31</sup> Although this damage to the DNA may be repaired, permanent cell transformation may occur if the repair mechanisms are ineffective or if the damage is not repaired correctly.<sup>32</sup> In the case of asbestos, the target cells that appear to be trans-

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<sup>27</sup>Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," p. 138.

<sup>28</sup>Ibid. See also, RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 16-17.

<sup>29</sup>Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," p. 138; and Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1449.

<sup>30</sup>Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1449.

<sup>31</sup>See generally, Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," pp. 1446-1455; John Cairns, *Cancer: Science and Society* (San Francisco: W.H. Freeman and Company, 1978); and U.S., Department of Labor, Occupational Safety and Health Administration, "Identification, Classification and Regulation of Potential Occupational Carcinogens," 29 CFR Part 1990, 45 FR 5002-5296, 22 January 1980 [hereafter "The OSHA Cancer Policy"].

<sup>32</sup>There is some suggestion in the scientific literature that carcinogenesis is not an irreversible process necessarily, so that even though the initial interaction with the DNA has taken place, the cells are capable of being repaired by a series of repair mechanisms available to the genetic material. RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 113-114; RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 92; and Cairns, *Cancer: Science and Society*, pp. 53, 85ff. However, it cannot be concluded that the repair mechanisms are completely reliable. Indeed, many authorities believe that DNA repair is not normally 100% efficient. See "The OSHA Cancer Policy," pp. 5124-5127.

Figure 4.1  
Photomicrograph of a Lung Showing the Changes Typical of Asbestosis\*



**Note:** \*Picture shows patchy fibrosis thickening some of the alveolar walls, while others remain normal, fine, and delicate.

**SOURCE:** Picture and letter from Dr. Alexander C. Ritchie, Professor of Pathology, University of Toronto to the Royal Commission on Asbestos, 4 October 1983.



formed are the epithelial cells of the bronchial tree and the gastrointestinal tract and the mesothelial cells of the pleura and peritoneum. There appears to be considerable evidence that at least some cancers may originate from a single transformed cell, but it is not entirely clear whether the asbestos-induced cancers fall into this category.<sup>33</sup> The early cellular and molecular events of the disease and the mechanisms which initiate it are not well understood.<sup>34</sup>

The asbestos-related cancers, like other cancers, are characterized by progressive cellular changes through a number of stages, leading to the development of abnormal or transformed populations of cells. Many cancer experts believe that a variety of different factors can act to initiate or accelerate the development of cancer at each stage and that often these factors will interact with each other, producing a synergistic effect.<sup>35</sup> That asbestos can act as a carcinogenic agent in its own right is strongly suggested by its effect in producing mesothelioma because the mesothelial tissues are not directly exposed to other environmental carcinogens.<sup>36</sup> That asbestos can also serve as a carcinogen by promoting or accelerating multiplication of malignant cells is strongly suggested by its effect in producing lung cancer in conjunction with cigarette smoking.<sup>37</sup> But whether asbestos fibres act in this way because of their physical dimensions, their inherent or acquired chemical composition and structure, or because of the effects of factors released or formed by their interaction with macrophages is again uncertain.

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<sup>33</sup> "The OSHA Cancer Policy," pp. 5023-5024; Kenny S. Crump et al., "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," *Cancer Research* 36 (September 1976): 2973; and Stephen Shugar, *Effects of Asbestos in the Canadian Environment*, prepared for the NRC Associate Committee on Scientific Criteria for Environmental Quality, NRCC, no. 16452 (Ottawa: National Research Council Canada, 1979), p. 134. If cancer can develop from a single cell, that cancer can be initiated by the interaction of a single molecule of a carcinogen with the critical target site in a cell. This does not necessarily mean that a person will develop cancer from the application of a single molecule of the carcinogen, since the probability of the interaction taking place appears to depend on the dose. Still, if a tumour can be traced back to a single molecular interaction, this suggests that any dose above zero may have an effect, and there is unlikely to be a threshold dose below which the induction of cancer cannot occur. See Chapter 5, Section E of this Report for a discussion of dose-response relationships.

<sup>34</sup> Craighead and Mossman have stated that "At present, the mechanism of asbestos-associated carcinogenesis is unclear, although the mineral appears to act like a classic tumor promoter. The fibrous nature of asbestos is critical . . ." See Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1452.

<sup>35</sup> "The OSHA Cancer Policy," pp. 5020-5022.

<sup>36</sup> Irving J. Selikoff and Douglas H.K. Lee, *Asbestos and Disease* (New York: Academic Press, 1978), pp. 417-423.

<sup>37</sup> *Ibid.* See also, Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1451.



Not only is there uncertainty as to how asbestos causes tumours at the target site, it is even uncertain as to how asbestos fibres get to the site of the tumour in the first place. In the case of pleural mesothelioma, it is thought likely that some of the asbestos deposited in the lung tissue reaches the pleura either by lymphatic transport within macrophages, or by the direct penetration of free fibres, or through the bloodstream.<sup>38</sup> Once positioned in the pleura, some of the fibres evidently are able to cause the development of mesothelioma, but the reasons for that development are not known. Similar uncertainty surrounds the development of peritoneal mesothelioma. It has been suggested by some authorities that asbestos fibres in the lungs are transported to the abdomen through the lymphatic channels of the body.<sup>39</sup> Once asbestos has entered the abdomen, it is thought that the mechanisms of carcinogenesis of the pleural and peritoneal cavities are similar.<sup>40</sup> But, again, the mechanisms of the malignant transformation of the mesothelial tissues remain obscure.

The same may be said for the development of asbestos-induced lung cancer. It was originally thought that this tumour was a scar cancer stimulated by the presence of asbestosis in the lungs because of its common occurrence in association with fibrosis and because of its common location in the lower lobes of the lung where fibrosis tends to be most marked.<sup>41</sup> However, reported cases of asbestos-induced lung cancer, either in association with very mild fibrosis or in its absence altogether, have left the reasons for the development even of this tumour unresolved.<sup>42</sup>

The way in which asbestos reaches the gastrointestinal tract so as to induce alimentary tract tumours is also far from firmly established. One possibility is that the coughing up and swallowing of fibres following in-

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<sup>38</sup>RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 78; John M.G. Davis, "The Biological Effects of Mineral Fibres," *Annals of Occupational Hygiene* 24:2 (1981): 228; and Personal communication between Professor Patrick Sebastien, Institute of Occupational Health and Safety, McGill University, Montreal, Quebec and Royal Commission on Asbestos Staff, 21 April 1983.

<sup>39</sup>Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1450.

<sup>40</sup>*Ibid.*

<sup>41</sup>RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, p. 77; and Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 214.

<sup>42</sup>Dr. J. Corbett McDonald testified before this Commission that his data are compatible with most excess lung cancers having occurred in people who had x-ray abnormalities. But he also indicated that there is evidence that some of the excess lung cancers he has observed are not explained in that way. In his view, there is no special reason to believe that the two processes of asbestosis and lung cancer are produced by the same mechanisms. RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 37. Dr. Becklake suggested there may be two different cell types of cancer attributable to asbestos: one which relates to squamous cell cancer in the major airways, and one which is a true scar cancer and therefore likely to be related to or stimulated by the presence of fibrosis in the lungs. RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, p. 77.

halation is responsible.<sup>43</sup> Another possibility, suggested by Dr. John M.G. Davis in his testimony before this Commission, is that asbestos fibres after inhalation have the ability to move around the body by the lymphatic channels to various sites, including the gastrointestinal tract.<sup>44</sup> Dr. Alexander C. Ritchie, Professor of Pathology at the University of Toronto, expressed serious reservations about this possibility in his testimony before this Commission. Dr. Ritchie had no doubt that fibres find their way to the gastrointestinal tract, but he candidly observed that we just do not know how they get there.<sup>45</sup>

The carcinogenic effects of asbestos do appear to have certain distinctive features in common with the effects of other carcinogens. For one thing, the effects of asbestos appear to be irreversible, not in the sense that cells transformed by contact with asbestos will inevitably develop into cancer, but that they will retain their potential to do so.<sup>46</sup> This means that humans exposed to asbestos will generally remain at increased risk of developing cancer even after exposure to asbestos has ceased. A second distinctive feature of asbestos-related cancers is that they are age-dependent.<sup>47</sup> Cancers in general tend to occur among older age groups, and asbestos-related cancers are no different.<sup>48</sup> A final feature that asbestos-related cancers share in common with other cancers is one we have already

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<sup>43</sup>Eric J. Chatfield, "Measurement of Asbestos Fibres in the Workplace and in the General Environment," in *Short Course in Mineralogical Techniques of Asbestos Determination*, p. 114; Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Asbestos Fibers," p. 138; and Anthony B. Miller, "Asbestos Fibre Dust and Gastrointestinal Malignancies," Appendix 5 in The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981.

<sup>44</sup>RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 100-101. Dr. Davis contrasted the results of animal inhalation experiments and animal ingestion experiments. Following the former he was able to find asbestos fibres scattered all around the animals' bodies, including the gastrointestinal tract; following the latter he could not find any asbestos in the gastrointestinal tissue of animals.

<sup>45</sup>RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, pp. 14-19, 31-35. Dr. Ritchie testified that in his opinion it was most improbable that asbestos fibres reached the gastrointestinal tissue by the lymphatics. He indicated that the lymph flow from the stomach to the lung is from the stomach upwards, so that one has to imagine the asbestos fibres swimming against the flow. Even if one allows that the fibres could proceed against the normal direction of flow, on the ordinary view that the dangerous fibres are the long, thin ones, then the fibres would be in little vessels that are really not much bigger than the fibres themselves and certainly much narrower than 10 microns in length. Dr. Ritchie himself preferred "the bloodstream to the lymph stream" as the more likely mechanism by which fibres are transported to the gastrointestinal system. But he went on to indicate that both the bloodstream and direct passage through the tissues seem to him to be improbable mechanisms. He did, however, testify that from two cases he had personally examined, he knew that asbestos fibres can be found in the tissues in and around gastrointestinal carcinomata.

<sup>46</sup>"The OSHA Cancer Policy," p. 5024.

<sup>47</sup>*Ibid.*, p. 5026.

<sup>48</sup>For a more detailed discussion of the age- and time-dependence of asbestos-related cancers, see Chapter 5, Section E of this Report.

observed in this Report: there is normally a latency period of many years between time of first exposure and the clinical manifestation of the disease.<sup>49</sup>

One final matter deserves mention. Whether any particular individual will develop a malignant tumour, the amount of exposure necessary for the initiation of carcinogenesis, and the rate of growth and spread of a tumour, once initiated, appear to depend upon a number of factors all of which may be subsumed under the term "individual susceptibility."<sup>50</sup> The efficiency of an individual's lung clearance mechanism, the anatomic characteristics of his lung and airway system, the physical fitness of the individual, and the nature of his immune system all suggest that some individuals are more susceptible to the development of cancer than others.<sup>51</sup> There is no concrete evidence which identifies individual susceptibility in relation to asbestos exposure and either cancer or fibrosis, but common sense and experience tell us that such individual susceptibility does indeed exist.<sup>52</sup>

### C. Principal Sources of Information on the Health Effects of Asbestos

The evidence on the etiology of asbestos-related diseases is derived chiefly from three sources: (i) the human experience, as measured by case reports and epidemiological studies of defined populations; and more limited post-mortem studies which analyze by electron microscopy the asbestos fibre content of lung tissue; (ii) experimental work with animals; and (iii) *in vitro* or cell culture studies.

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<sup>49</sup>"The OSHA Cancer Policy," p. 5026. But the traditional theory of latency, at least in respect of asbestos-related diseases, has recently been challenged. See Chapter 5, Section E of this Report.

<sup>50</sup>RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, pp. 40-41. However, Dr. Ritchie knew of no evidence which identifies susceptibility in relation to asbestos exposure and cancer. See also, Philip E. Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," *Journal of Occupational Medicine* 18:3 (March 1976): 150-151; and "The OSHA Cancer Policy," pp. 5129-5138.

<sup>51</sup>Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," pp. 190-191.

<sup>52</sup>Dr. William J. Nicholson testified that the Mount Sinai School of Medicine in New York is beginning to look at the possibilities of identifying individuals at particularly high risk among those equally exposed; in other words, what means exist to predict who would be likely to get cancer and who would not. The possibility exists that this can be done by measurement of immune competence. RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 134. See also, Elliott Kagan, "Immunological Status and Host Resistance to Asbestos-Related Disease," in *Proceedings of the World Symposium on Asbestos*, Montreal, Quebec: 25-27 May 1982 (Montreal, P.Q.: Canadian Asbestos Information Centre [1983]), pp. 442-448. The author indicated that research is being done in the area of sophisticated immunological probes to detect persons "at risk" of developing asbestos-related cancers.



Given the uncertainty that exists about many of the most important health issues concerning asbestos, the available evidence from the various studies and tests requires careful assessment. That assessment can best be done within the framework of an accepted set of principles that will enable us to interpret and weigh properly the health evidence before us. For such a set of principles, we again turn to Dr. Sackett and to the tests for causation articulated by him.<sup>53</sup> Those tests particularly relevant to studies of the association between asbestos exposure and disease (listed in descending order of importance) are as follows:

- (i) Is there a strong association between exposure and health outcomes of interest? "Strong" here means the odds favouring the outcome of interest with, as opposed to without, exposure to asbestos. The weaker the association, the greater the need for caution in relying upon the result. A fivefold increase in the incidence of lung cancer among an asbestos-exposed population would be strong evidence of a relationship between asbestos exposure and the disease. A twofold increase would have to be treated more cautiously.
- (ii) Is the association consistent from study to study? The repetitive demonstration by different investigators studying different populations of an association between exposure and the outcome of interest gives consistency and thus credibility to the association.<sup>54</sup> For example, much of the credibility concerning the causal link between smoking and lung cancer arises from the repeated demonstrations of a strong statistical association in case-control, cohort, and other studies.
- (iii) Is there a dose-response relationship? As it is an accepted biological principle that the greater the dose the greater the response, studies which demonstrate not simply a response but also a response that increases with dose provide greater support for a causal relationship. This is a crucial issue and is considered at length in Chapter 5 of this Report.
- (iv) Does the association make epidemiological sense? Are the investigator's results in agreement with current understanding of the distribution of causes and health outcomes in humans?

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<sup>53</sup>Sackett, "A Review of Diagnostic Tests for Causation," pp. 10-16 and Appendix B, *passim*. These tests were originally articulated by Austin Bradford Hill, *Principles of Medical Statistics*, 9th ed. (London, England: Oxford University Press, 1971), p. 320. See also, J. Corbett McDonald, "Asbestos and Lung Cancer: Has the Case Been Proven?" *Chest* 78:2 (August 1980, Supplement): 374-376; and Austin Bradford Hill, *A Short Text-book of Medical Statistics* (London, England: Hodder and Stoughton, 1977), pp. 285-296.

<sup>54</sup>RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, p. 84.



(v) Does the association make biological sense? Is it consistent with current understanding of the responses of human cells, tissues, and organs to the postulated causative agent or agents?

(vi) Is the association specific? Is the association limited to a single putative cause and a single effect, or is the association clouded by the possibility of multiple causes and overlapping effects? Asbestosis and mesothelioma have a quite specific association with asbestos exposure. On the other hand, lung, laryngeal, and gastrointestinal cancer, which can be associated with asbestos exposure, are also associated with smoking, diet, and lifestyle, thus clouding the association.

(vii) Is the association analogous to a previously proven causal relationship? This test links, for example, asbestosis among asbestos workers who inhale dust particles containing asbestos to the earlier known causal relationship between silicosis and the inhalation of dust particles containing silica.

Not all of these tests are applicable to every health issue before us, nor need each test be satisfied in order to demonstrate a particular causal relationship. Rather, proof of an association will depend upon the weight of the available evidence.

## **D. The Human Experience: Epidemiological Studies**

Epidemiology is concerned with the study of the distribution and determinants of disease prevalence in humans. Asbestos has been the subject of a number of epidemiological studies which are reviewed in detail in Chapter 5 of this Report. The overwhelming importance of epidemiological data is that they provide evidence in humans of associations between exposures to various substances and patterns of disease. Specifically — and to the extent the necessary data are available — they permit the direct measurement of the risk of cancer or fibrosis due to exposure to asbestos in human populations.

The true experiment with humans, the so-called randomized trial, is the optimal epidemiological method to explore questions of causation. In the case of asbestos, the randomized trial is neither feasible nor ethical. This rules out a true experiment that would deliberately expose a randomly selected group of individuals to given doses of asbestos and compare their health outcomes to a randomly selected control group. Three other epidemiological methods are, however, available to examine the human disease experience with asbestos: the cohort study, the case-control study,

and the case-series study. Of these, the most powerful analytical technique is the cohort study.<sup>55</sup>

## D.1 The Cohort Study

The essential design of a cohort study is to define a group of persons exposed to a particular hazard (the cohort) and a group not so exposed (the control group). The experience of these two groups is followed over time and, where appropriate records are available, the morbidity and mortality experiences of the two groups are compared.

Typically, cohort studies may be retrospective or prospective. The cohort studies of asbestos-exposed populations published to date have been retrospective, so that what is being assessed is the past health experience of a group of workers exposed to asbestos. The fact that asbestos cohort studies are retrospective may impose limitations on their quality in that the epidemiologist can only use what is available in terms of exposure data, work histories, and the like. An asbestos mortality study might attempt to compare the observed number of deaths attributable to lung cancer in the selected cohort with the expected number of deaths due to lung cancer in the control group. The calculation of the expected number of deaths is made by reference to a more general population base, assumed not to be exposed to asbestos, be it the country as a whole, or the province or state where the cohort is located. This calculation of the expected number of deaths is standardized to take account of the death rates in specific age groups and in specific calendar time periods. The epidemiologist may also control for sex, race, and other factors to make the comparison more precise. In the result, the epidemiologist obtains a ratio of the observed number of deaths in the cohort to the expected number of deaths. This ratio is known as a standard mortality ratio (SMR), which is a measure of relative risk. An observed/expected ratio for lung cancer of 2, or an SMR of 200, means that the actual deaths from lung cancer in the cohort exposed to asbestos is twice the number that may be expected in the ordinary population not exposed to asbestos. An alternate way of saying the same thing is that the excess risk of lung cancer in the cohort is 100%. Studies which attribute death to mesothelioma or asbestosis do not generally provide an expected number of deaths calculation for the simple reason that deaths from these two causes are so rare in the general population (or control group) as to be zero for practical purposes.

Most asbestos cohort studies provide some quantitative data on exposures, in order that observed/expected ratios may be calculated at different exposure levels. Such information is necessary to determine dose-

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<sup>55</sup>Sackett, "A Review of Diagnostic Tests for Causation," pp. 11-12 and Appendix B, pp. 2-4.

response relationships and to assess the incidence of disease in the cohort in terms of the severity of exposure. The common method of quantifying asbestos exposure in order to assess health risks has been to calculate exposures both in terms of the concentration at a given time and duration of exposure over time. The result, a measure of cumulative exposure, has been expressed in either million particles per cubic foot-years (mppcf-yrs) or fibres per cubic centimetre-years (f/cc-yrs), depending upon whether what has been measured were particles of dust or asbestos fibres. One fibre per cubic centimetre-years represents exposure to an average workday concentration of 1 fibre per cubic centimetre (f/cc) for 1 year; 2 fibres per cubic centimetre-years would represent either 2 years of exposure at 1 f/cc or 1 year of exposure at 2 f/cc. Similarly, 8 fibres per cubic centimetre-years might represent 1 year of exposure at 8 f/cc, 2 years of exposure at 4 f/cc, or 4 years of exposure at 2 f/cc. Where a cohort study does not have available quantitative measurements, duration of exposure is generally used as a surrogate for cumulative dose.

There is in cohort analysis the possibility of sampling variation or random error in measurement, which raises concerns that the standard mortality ratio (or relative risk) may indicate that there is an adverse health effect when in truth there is not; or may indicate that there is no effect when in truth there is. Accordingly, authors of most of the epidemiological studies have provided statistical confidence intervals to their calculations and have indicated where a calculation is statistically significant as opposed to being the possible result of chance.

## **D.2 Criteria for Evaluating Cohort Studies and the Limitations of Asbestos Epidemiology**

Just as there exists a set of criteria for the general evaluation of health effects evidence, there is a separate set of recognized criteria for assessing the quality of cohort studies. Several of our expert witnesses made reference to these criteria during the course of their testimony.<sup>56</sup> We deem it useful to make reference to them in our Report for they both provide a framework for the critical appraisal of the epidemiological literature and enable us to focus upon the deficiencies in asbestos epidemiology. However unavoidable these deficiencies are, given the data that are available, their expression serves to emphasize the uncertainty that pervades the evidence on the health effects of asbestos.

For convenience, we group the relevant criteria under four headings: the study population; the quantification of exposure; mortality or morbidity ascertainment; and statistical analysis.

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<sup>56</sup>For a general discussion of these criteria, see the Appendix to Chapter 7 of this Report.



### (a) The Study Population

*Selection Criteria* — To ensure that any effects of exposure will manifest themselves, a sufficient number of the study population should have a substantial amount of exposure. The study by Dr. J. Corbett McDonald et al. of the Quebec chrysotile miners and millers is likely the best in this regard for the authors identified everyone who had ever worked for one month or more in the mines and mills and who was born between 1891 and 1920.<sup>57</sup> We note that it may be disadvantageous to include only long-term employees because they may be a survivor population and hence may not give a true picture of the disease incidence in the cohort. The study by Dr. Philip E. Enterline and Ms. Vivian L. Henderson of Manville employees has been criticized because the authors selected only retirees, those over age 65, for inclusion in their cohort. On the other hand, as Dr. Enterline pointed out, there is an advantage to studying retirees. Since they are no longer being exposed, one can easily separate the effects of dose from the effects of duration of exposure.<sup>58</sup> Ideally, the epidemiologist would wish an even distribution of employees in terms of age, duration of exposure, and intensity of exposure. In practical terms, he can do no better than the actual distribution of the cohort he is studying.

*Number of Subjects* — The capacity of a morbidity or mortality study to detect excess risk is directly related to the size of the cohort and to the observed incidence of disease or number of deaths. A small cohort and a small excess risk may not demonstrate a statistically significant association.<sup>59</sup> The epidemiologist thus has to ensure an adequate number of individuals for study within the cohort to be able to detect with reasonable statistical confidence a true increase in the level of disease. The Quebec study of miners and millers (with over 11,000 members of the cohort and over 4,500 observed deaths), the Ferodo friction materials plant study (with over 13,000 members of the cohort and over 2,100 deaths), and the North

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<sup>57</sup>J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," *British Journal of Industrial Medicine* 37 (1980): 11-24.

<sup>58</sup>Philip E. Enterline and Vivian L. Henderson, "Type of Asbestos and Respiratory Cancer in the Asbestos Industry," *Archives of Environmental Health* 27:5 (November 1973): 316-317; and RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 38-40. For examples of criticisms of the use of a survivor population, see RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 30; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 83-85.

<sup>59</sup>See, for example, RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, pp. 32-34.



American insulators study (with 17,800 members of the cohort and over 2,200 deaths) offer potentially the strongest evidence.<sup>60</sup>

Of course, the maximum cohort size is invariably fixed by the number of past employees so that the investigator is bound by the available population and there are few large cohorts of asbestos workers. This gives rise to a serious limitation of asbestos epidemiology: it is likely not sensitive enough to detect excess risk directly or without extrapolation at the lower levels of exposure that currently prevail — that is, 2 f/cc and below — unless the effect is a very large one. According to Dr. Enterline, even if the excess risk at 2 f/cc is as high as 25%, meaning that the SMR is 125, that excess is not statistically detectable by epidemiological methods for the simple reason that a large enough study population is not available.<sup>61</sup>

*Type of Asbestos Exposure* — There may be no asbestos-related health issue more vexing or more important than whether different fibre types or dimensions differ in their health effects. The investigation of this issue can be facilitated in a situation where the entire study population has been exposed to a single fibre type and in a type of process likely to yield fibres of a given size. The study by Dr. J. Corbett McDonald et al. of the miners and millers in Quebec (chrysotile only); the study by Mr. Geoffrey Berry and Dr. Muriel L. Newhouse of the Ferodo friction materials factory workers at Derbyshire, England (chrysotile only); the study by Dr. John M. Dement et al. of the asbestos textile factory workers at Charleston, South Carolina (chrysotile only); and the studies of the war-time gas mask workers by Dr. Alison D. McDonald and Dr. J. Corbett McDonald in Canada (crocidolite only) and by Dr. J.S.P. Jones et al. in England (separate crocidolite and chrysotile exposures) are examples of studies that

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<sup>60</sup>J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75"; Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 1-7; and Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91-116.

<sup>61</sup>Ontario, Royal Commission on Asbestos, Exhibit II-3 [hereafter RCA Exhibit], in RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8: Philip E. Enterline, "Epidemiologic Basis for the Asbestos Standard," paper presented at the Second Annual Symposium on Environmental Epidemiology, Pittsburgh, Pennsylvania, 28 April 1981, Table 2. (Mimeographed.) See also, RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 26-33. Dr. Enterline addressed the question as to whether epidemiology can provide any evidence on the safety of the 2 f/cc standard. In his testimony, he offered the view that epidemiology might detect an excess risk of 50% or greater. Thus, for example, Dr. J.C. McDonald's study of the Quebec miners indicated a 6% excess risk after 50 years' exposure at 2 f/cc. According to Dr. Enterline, a study population of 220,000 would be required for that excess to be statistically detectable.

best meet this criterion.<sup>62</sup> The investigation of this issue can also be facilitated in a situation where a cohort can be divided into groups of employees whose exposure differed only in fibre type, fibre size, or type of process, thereby providing an internal comparison within the same working environment. The Enterline study is of some assistance here, in that part of his cohort was exposed to single fibre types and part to a combination of chrysotile and the amphiboles.<sup>63</sup> If, however, the cohort being studied has had a mixed fibre exposure as, for example, in the study by Dr. Murray M. Finkelstein of the asbestos-cement workers at Scarborough, Ontario,<sup>64</sup> it makes inquiry into the possible differential pathogenicity of different fibre types and fibre sizes that much more difficult. Unfortunately, few study populations or groups within study populations have been exposed solely to one fibre type, size, and process. Given that these factors may affect the disease rate, in a situation where these factors differ as between studies, it is difficult to sort out the most salient features.

### (b) The Quantification of Exposure

In light of our discussion thus far, the need for quantitative data on exposure levels is obvious. Here, three main factors come into play. First we list them and then discuss their limitations. A fourth factor, smoking data, is considered separately.

*Work History* — It is important that the epidemiologist have an accurate description of each individual's job history and where in the plant being studied each job was performed. Only then can he hope to utilize the measurements available for the plant in order to determine each cohort member's overall exposure.

*The Quantity of Industrial Hygiene Data* — This criterion relates to the number of measurements taken in the plant, where in the plant they were taken, and the years in which they were taken. In the absence of actual

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<sup>62</sup>J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75"; Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos"; John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1-4 (1982): 869-887; Alison D. McDonald and J. Corbett McDonald, "Mesothelioma After Crocidolite Exposure During Gas Mask Manufacture," *Environmental Research* 17 (December 1978): 340-346; and J.S.P. Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 637-653.

<sup>63</sup>Philip E. Enterline, Pierre DeCoulfe, and Vivian L. Henderson, "Mortality in Relation to Occupational Exposure in the Asbestos Industry," *Journal of Occupational Medicine* 14:12 (December 1972): 900; and Vivian L. Henderson and Philip E. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 122.

<sup>64</sup>Murray M. Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," *British Journal of Industrial Medicine* 40 (1983): 138-144.

measurements, duration of exposure, which does not differentiate as to the level of exposure, must serve as an index of dose.

*The Quality of Industrial Hygiene Data* — The quality of the data in asbestos epidemiology is basically related to questions of whether measurements were made in fibres or in dust particles, whether there were any side-by-side measurements, and whether there is any reliable relationship between particle counts and fibre counts in the cohort under review.

As we have indicated, cohort studies of asbestos workers by their nature are historical or retrospective rather than prospective: the studies were all undertaken long after the exposures occurred. The epidemiologist must work backwards in time to gather the necessary data for his study. It is therefore no accident that one of the weakest parts of all asbestos epidemiology is the quality of the quantitative exposure data. Although asbestos is perhaps the best studied carcinogen, next to radiation, the industrial hygiene data are seriously deficient.<sup>65</sup> In particular, we identify the following deficiencies:

(i) Work histories tend to be inaccurate because industry kept very little by way of detailed records. Even where work histories are available, the description of the position a worker held does not necessarily divulge what he did. Such exposure data as do exist are not refined so that there is little or no evidence on where an employee was in a plant and what hours of the day he was there, data which could be very important in determining his real exposure.<sup>66</sup>

(ii) There is a serious lack of measurements of any kind, particularly in the period before 1950. Those are precisely the years when exposures were higher and when by now a sufficient period has elapsed so that the disease effects of that exposure have become manifest. For example, Dr. Dement's study had over 5,000 individual measurements in the plant during the period 1960–1975, but less than 200 measurements in each of the periods 1930–1945 and 1945–1960.<sup>67</sup> While Dr. J.C. McDonald, in his Quebec study, had over 4,000 midget impinger readings available in the period 1949–1966, very few of these were in underground mines or open pits where the bulk of the cohort was exposed and there were virtually no readings of

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<sup>65</sup>RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 90.

<sup>66</sup>See, for example, Julian Peto, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part II. Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 197. See also, the Appendix to Chapter 7 of this Report.

<sup>67</sup>Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," Table 1, p. 871.



any kind in the pre-1949 period.<sup>68</sup> In the Scarborough plant studied by Dr. Finkelstein, measurement data exist for only four of the years between 1948 and 1960.<sup>69</sup>

(iii) Almost all of the measurements in the cohort studies now available were in terms of dust particles, whereas exposure to asbestos is now regulated in terms of fibres. This would not necessarily be a weakness if there was a reliable and accurate method of converting particle measurements to fibre measurements. Considerable efforts have been made by several epidemiologists to convert the dust measurements in their studies to fibre counts. On the basis of the testimony we heard concerning the results of these efforts, we can say generally that converting dust particles to fibres is an uncertain process.<sup>70</sup> Moreover, the conversion factors that apply, for example, in textiles are likely not applicable to those in mining or even in friction materials. Even the conversion factors for textile carding and spin-

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<sup>68</sup>J. Corbett McDonald and F. Douglas K. Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 1-9; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 5; and RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 31. See also, the Appendix to Chapter 7 of this Report.

<sup>69</sup>Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory"; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 46-48.

<sup>70</sup>The process is sufficiently uncertain that, in their earlier publications, Dr. J.C. McDonald and his colleagues indicated that they could not express their results confidently in fibre counts. See J. Corbett McDonald et al., "The Health of Chrysotile Asbestos Mine and Mill Workers of Quebec," *Archives of Environmental Health* 28:2 (February 1974): 68. In their later work, Dr. J.C. McDonald et al. attempted to convert dust particle measurements from the Quebec mines to fibre measurements. In his oral testimony before this Commission, Dr. J.C. McDonald commented on their efforts as follows:

It's more or less fizzling out, actually. I mean, we're beginning to get depressed about it. We've been doing it for a good five or six years, and I think we know how almost unanswerable the problem is. (RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 42.)

Dr. Graham W. Gibbs and Dr. Maurice Lachance in 1971 and 1972 conducted surveys in each of the five mines and mills in the Quebec chrysotile mining and milling industry. A total of 87 pairs of membrane filter samples and midget impinger samples were collected side by side at each location. The results did not satisfactorily provide a prediction of fibre counts from impinger counts, and the authors concluded that until satisfactory conversion factors were derived it would be preferable to base standards for at least mines and mills on particle counts for which at the time there existed a reasonable body of evidence. See Graham W. Gibbs and Maurice Lachance, "Dust-Fiber Relationships in the Quebec Chrysotile Industry," *Archives of Environmental Health* 28:2 (February 1974): 69-71. Dr. Kenny S. Crump suggested in his evidence that the correlation between fibre counts and dust counts is less the lower the exposure levels. See RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, pp. 17-19.



ning do not likely apply to other textile operations. Within a specific process, conversions may be fairly reliable.<sup>71</sup>

(iv) There is likely considerable inaccuracy or at least variability in the measurements that do exist. This adds to the unreliability of the exposure data from the cohort being studied. It also raises concerns about comparing the results of different cohort studies. Even when counted by a trained operator, some variability in the measurement of the same sample may occur;<sup>72</sup> this effect is doubtless compounded when the operator is not qualified, as had often been the case in the years covered by the historical studies. Improving technology and changing methodology of measurement over time represent a further source of variability.<sup>73</sup> For example, if one were to go back in time using today's technology and methodology to measure the same dust cloud, undoubtedly one could produce a higher figure now than was originally counted.<sup>74</sup>

(v) Even where measurement data exist there is no information on the size distributions of fibre concentrations. There is also no information on climatic conditions that prevailed in the various plants being studied (for example, whether conditions were dry or humid). These conditions may have affected fibre levels and fibre size distributions.

(vi) There are no measurements at the low end of the dose-response curve, which is the area of current and future concern. It is only in the last decade or so that industry exposures have been reduced to current levels. Accordingly, we have no measurements at or below 2 f/cc sufficiently far back in time to enable us to look at the actual effect of these levels. In order to

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<sup>71</sup>RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 60; RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 112; Gibbs and Lachance, "Dust-Fiber Relationships in the Quebec Chrysotile Industry"; RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 44-45; and Yehia Y. Hammad, John Diem, and Hans Weill, "Evaluation of Dust Exposure in Asbestos Cement Manufacturing Operations," *American Industrial Hygiene Association Journal* 40:6 (June 1979): 490-495.

<sup>72</sup>RCA Transcript, Evidence of Dr. Graham W. Gibbs, 13 November 1981, Volume no. 33, p. 39; Graham W. Gibbs et al., "A Summary of Asbestos Fibre Counting Experience in Seven Countries," *Annals of Occupational Hygiene* 20:4 (December 1977): 321-332; G.W. Gibbs, R.S. McCullough, and R.A. Tate, "A Canadian Asbestos Counting Trial," *Canadian Journal of Public Health* 70 (September/October 1979): 343-350; and RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 119.

<sup>73</sup>RCA Transcript, Evidence of Dr. Gerald R. Chase and Dr. Harrison B. Rhodes, 14 August 1981, Volume no. 27, p. 76. See also, RCA Exhibit II-27, Tab 19, in RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18; Eric J. Chatfield, "Airborne Asbestos Fibres: A Summary of Some Measurement Problems," Sheridan Park, Ontario Research Foundation, 1981, p. 3. (Mimeographed.)

<sup>74</sup>RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 18; and RCA Transcript, Evidence of Dr. Gerald R. Chase and Dr. Harrison B. Rhodes, 14 August 1981, Volume no. 27, p. 76.

attempt any assessment of the disease risk at such levels, we are therefore compelled to extrapolate beyond actual observation.<sup>75</sup>

(vii) There is a lack of necessary individual data to provide a suitable base to compare the effects of short, intermittent, high exposures with continuous but lower exposures. In other words, the exposure data that do exist do not distinguish which individuals received their dose at a constant level and which individuals received their dose in shorter but more intense bursts.<sup>76</sup>

*Smoking Data* — The availability of information on the smoking habits of the cohort is important at least in terms of the incidence of lung and laryngeal cancer, where smoking is a contributing factor.<sup>77</sup> Such information generally has to be gathered by a personal interview or questionnaire of employees, or if deceased, of relatives. Frequently such information either is not available or has not been gathered by the investigator. Where it is available, it is often incomplete and is in any event of questionable reliability, as people are not always frank as to their true smoking habits.<sup>78</sup>

### (c) Mortality or Morbidity Ascertainment

*Completeness of Follow-up* — This criterion relates to whether the epidemiologist has been able to trace his entire study population; that is, to ascertain what has actually happened to each member of that population. If his ascertainment rate is low, then one need be concerned as to whether the persons not traced are more likely to have died of asbestos-related disease than those accounted for, in which case even the level of mortality indicated by the study would underestimate the true mortality. Of course, such a bias could also work the other way, and there is generally no reason to expect a relationship between ascertainment rate and cause of mortality (or morbidity).<sup>79</sup> However, the fact remains that with less than a 100% ascertainment rate, there is bound to be uncertainty as to the overall level of mortality and potential for bias in the results. Fortunately, virtually all of the main epidemiological studies have trace rates of 90% or above. The exception is the published study by Weill, Hughes, and Waggenpack of the New Orleans

<sup>75</sup>Sackett, "A Review of Diagnostic Tests for Causation," p. 15 and Appendix B, p. 8.

<sup>76</sup>Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," p. 154; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 62; and RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, p. 121. Dr. A.D. McDonald testified that "... epidemiologically we have absolutely no means for distinguishing between the short, sharp or the continuous, low [dose]. . . ."

<sup>77</sup>See the discussion of "the effect of smoking" in Chapter 5, Section F of this Report.

<sup>78</sup>See the Appendix to Chapter 7 of this Report.

<sup>79</sup>RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 68-70; and RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 65.

asbestos-cement workers, where the trace rate is only 75%.<sup>80</sup> (However, Dr. Hans Weill is in the course of updating this study and has achieved a much higher ascertainment rate.)<sup>81</sup>

*End-Point Ascertainment* — This criterion refers to the accuracy of the diagnosis of the diseases identified in the cohort and the availability of death certificates for all deaths. If the disease is identified on the basis of a death certificate (which is the usual method for determining cause of death), the epidemiologist must consider whether it is reasonable to accept the information there stated or whether to use additional information (from autopsy reports, for example) to confirm or, if necessary, correct the diagnosis. We have been told that death certificates are of limited reliability in determining whether death was caused by an asbestos-related disease.<sup>82</sup> This was particularly true for those deaths years ago when diseases from asbestos exposure were not as well known to the ordinary practising physician and pathologist and when, as a result, the possibility of misdiagnosis in favour of other better-known diseases was considerable.<sup>83</sup> Asbestosis and mesothelioma are in any event open to misdiagnosis if only because they are not often seen by physicians in the general practice of medicine.

It is noteworthy that for the purposes of international diagnostic classification, mesothelioma of the pleura and the peritoneum were not even assumed to be malignant until the Eighth Revision of the International

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<sup>80</sup>Hans Weill, Janet Hughes, and Carmel Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease* 120:2 (August 1979): 345-354.

<sup>81</sup>Letter from Dr. Hans Weill, Pulmonary Diseases Section, Department of Medicine, Tulane Medical Center, New Orleans, Louisiana to the Royal Commission on Asbestos, 11 April 1983.

<sup>82</sup>It appears that death certificates are apt to be particularly unreliable in the case of mesothelioma. Dr. Kotin has written that the likelihood of incorrectly diagnosing mesothelioma is so great that review of diagnoses of mesothelioma by panels of experts is recognized as indispensable throughout the world. He has also noted that even among the experts from the United States Mesothelioma Panel, there is at best agreement on the diagnosis in only 80% of the cases under review. See RCA Exhibit II-30, Tab 9, in RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A): Johns-Manville Canada Inc., Written submission to the Royal Commission on Asbestos, prepared by Paul Kotin, M.D., #18, 15 January 1981, p. 9. See also, Alison D. McDonald, "Mesothelioma Registries in Identifying Asbestos Hazards," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 444-445; RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, pp. 92-93; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 85-87. Dr. Ritchie testified that pathologically mesothelioma is still a difficult diagnosis. Dr. Weill gave evidence that for a long period of time there had been an under-diagnosis of mesothelioma. He went on to say that mesothelioma continues to be a difficult diagnosis and that even the mesothelioma panels now operating in Canada, the United States, and Great Britain have not greatly reduced inter-observer variability in the diagnosis of this tumour. Dr. Weill also pointed out that some authorities have suggested that there is now an over-diagnosis of mesothelioma.

<sup>83</sup>RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 85-87; and A.D. McDonald, "Mesothelioma Registries in Identifying Asbestos Hazards," pp. 441-443.



Classification of Diseases (ICD) in 1965 and were not automatically considered so until the Ninth Revision in 1975. Malignant neoplasm of the pleura first received a separate classification in the Eighth Revision, and peritoneal mesothelioma was not adequately delineated until the Ninth Revision.<sup>84</sup> Despite the increasing awareness of the effects of asbestos exposure, and despite increasing medical knowledge of asbestos-related disease, there remains serious inaccuracy in the certification of this cause of death. Dr. J. Corbett McDonald and Dr. Alison D. McDonald, who have extensively investigated the etiology of mesothelioma, have estimated that inaccuracy may be as high as 50%.<sup>85</sup> By way of example, Dr. A.D. McDonald cited the fact that in the province of Quebec, one-half of all mesothelioma cases registered through pathologists could not have been identified from death certificates.<sup>86</sup> Given this inaccuracy, and given the fact that the incidence of

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<sup>84</sup>A.D. McDonald, "Mesothelioma Registries in Identifying Asbestos Hazards," pp. 444-445. In the Eighth Revision of the ICD, Code 163 is for "malignant neoplasms of other and unspecified respiratory organs." Code 163.0 is for "malignant neoplasms of pleura." In the Ninth Revision, Code 163 is for "malignant neoplasm of pleura" with the following subheadings: 163.0, parietal; 163.1, visceral; 163.8, other; and 163.9, pleura, unspecified. Pursuant to the Seventh Revision of the ICD, a number of pleural mesotheliomas were coded to 212, "benign neoplasms of the respiratory system," or, if the site of the tumour was not specified on the death certificate, to the classification for "benign neoplasms of the connective tissue" or "benign neoplasms of other and unspecified sites."

In the Eighth Revision, Code 158 is for malignant neoplasms of the peritoneum and retroperitoneum tissue. The only subheadings are Code 158.0 for retroperitoneal tissue and Code 158.9 for other parts. In the Ninth Revision, Codes 158 and 158.0 remained as in the Eighth Revision. However, there was a further breakdown and Code 158.8 was added for "specified parts of the peritoneum," while Code 158.9 is used for the "peritoneum, unspecified." Most of the neoplasms coded to 158.9 are peritoneal mesotheliomas. See *Eighth Revision International Classification of Diseases, Adapted for Use in the United States*, Public Health Service Publication, no. 1693 (Washington, D.C.: U.S. Government Printing Office, 1968); *International Classification of Diseases, 1975 [Ninth] Revision* (Geneva: World Health Organization, 1977); and *Comparability of Mortality Statistics for the Seventh and Eighth Revisions of the International Classification of Diseases, United States*, Department of Health, Education and Welfare Publication, no. (HRA) 76-1340, Vital and Health Statistics Series 2, no. 66 (Rockville, Maryland: U.S. Department of Health, Education and Welfare, October 1975); and Telephone communication between Mrs. Marian Heid, Nosologist, U.S. National Cancer Institute, Bethesda, Maryland and Royal Commission on Asbestos Staff, 8 November 1983.

<sup>85</sup>J. Corbett McDonald and Alison D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," *Preventive Medicine* 6:3 (September 1977): 432-433.

<sup>86</sup>A.D. McDonald, "Mesothelioma Registries in Identifying Asbestos Hazards," pp. 444-445. Dr. J.C. McDonald and Dr. A.D. McDonald have suggested that Quebec pathologists in general have over-diagnosed mesothelioma. They indicated that only 37% of cases examined from Quebec were accepted by the Mesothelioma Panel of the Canadian Tumour Reference Centre as compared with about 60% from other regions. Overall, they have concluded that the true rate of recognized mesothelioma is probably generally somewhat higher (perhaps by 50% or more) than that obtained through pathologists. J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," pp. 430-433. Dr. Nicholson testified that in his opinion approximately 30% of death certificates are incorrect in respect of mesothelioma. He suggested, for example, that cancer of the pancreas happens to be a common diagnosis for peritoneal mesothelioma on death certificates. RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 12-14.



mesothelioma or indeed asbestosis in the general population is minimal, there is at least some justification to go behind the death certificate and search for the best available evidence as to the true cause of death.<sup>87</sup> However, supplementing death certificate information with other information and in effect basing the cause of death in the study population on information different from that available for the control population runs the risk of biasing any comparisons that are made.<sup>88</sup>

*Length of Follow-up* — The period between the time of first exposure of the last entrants into the cohort and the date upon which the investigator has undertaken his study must be sufficiently long to ensure that the diseases of interest will have time to manifest themselves. Bearing in mind the latency period before the appearance of malignant tumours or clinical fibrosis, most of the studies have a minimum follow-up period of at least 15 years.<sup>89</sup> There are, however, exceptions and, for example, there is no minimum follow-up period in the study by Thomas et al. of asbestocement workers at Cardiff<sup>90</sup> and only a 10-year minimum follow-up period in the Berry and Newhouse study of the Ferodo plant.<sup>91</sup>

#### (d) Statistical Analysis

*Suitability of External Comparison Group* — Ideally, the epidemiologist would want to construct a comparison population or control group out of the very type of cohort being studied. If he was studying insulation workers exposed to asbestos, his optimal control group would be insulation workers not so exposed. However, the data to do this simply do not exist and accordingly most investigators are obliged to utilize local, province- or state-wide, or more usually, national population bases. When the national population provides the comparison group, there may be problems occasioned by the fact that the cohort reflects a mortality experience peculiar to the particular region in which the study was conducted or by reason of factors that are peculiar to, or characteristic of, working populations but are not evident in the general population.<sup>92</sup> On the other hand, if province,

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<sup>87</sup>RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 12-13; and Selikoff, Hammond, and Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," p. 114.

<sup>88</sup>Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," p. 152.

<sup>89</sup>See Chapter 5, Section B of this Report for a discussion of the major studies.

<sup>90</sup>H.F. Thomas et al., "Further Follow-up Study of Workers from an Asbestos Cement Factory," *British Journal of Industrial Medicine* 39:3 (August 1982): 273-276.

<sup>91</sup>Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos."

<sup>92</sup>See the discussion of this issue in Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," p. 153.

state, or other local population mortality data are used, they may be overly contaminated by the very cohort being studied.<sup>93</sup>

Occupational groups tend to be healthier and thus exhibit lower mortality from all causes than the general population. This fact, the so-called "healthy worker effect," means that when general population mortality data are used for the purpose of external comparison, it may underestimate the magnitude of the health effect of the substance of interest.<sup>94</sup> However, both Dr. J. Corbett McDonald and Mr. Geoffrey Berry, in their evidence before this Commission, indicated that while the healthy worker effect indeed biases some occupational studies, it is less likely to be a significant factor in the cases of diseases of long latency which affect older workers, like cancer or fibrosis; and from their testimony it would seem that the effect disappears after 15 years.<sup>95</sup>

*Internal Comparison* — Some cohort studies provide a separate internal comparison of certain aspects of the study, usually by a case-control analysis. The basic methodology in a case-control analysis is discussed below. Such an analysis may be used in conjunction with a cohort study to facilitate an investigation of the relationship between smoking, asbestos exposure, and lung cancer or to explore the influence of fibre type on the incidence of mesothelioma in the cohort. To the extent that such internal comparisons are made, they are helpful in supplementing and confirming the findings of the main cohort study, thereby adding to its reliability. Dr. J. Corbett McDonald's studies in Quebec and Mr. Julian Peto's recent

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<sup>93</sup>Dr. Dement has come under some criticism for his use of national mortality rates. Since the local counties in and around Charleston have a very high lung cancer rate — approximately 75% above the national average — had Dr. Dement compared with local rates, the incidence of excess disease would have been less. Dr. Dement rejected the use of local rates for comparison because of the effect that the plant being studied may have had on local lung cancer death rates and because of the likely increases in those rates due to the local shipyard industry. Even so, the rates for the contiguous counties would have increased expected lung cancer rates only by 15% and the state rate was close to the national rate. See Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Workers," pp. 879-880.

<sup>94</sup>Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," p. 153.

<sup>95</sup>RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 138; and RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, p. 69. See also, RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 85; and Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," p. 153.

study at Rochdale, England, have made particularly effective use of this methodology.<sup>96</sup>

*Time Since First Exposure* — All authors are aware of the need to allow an appropriately long latency period in calculating the disease risks of asbestos, and all studies calculate disease risks beginning at least 10 years (and more often 15 or 20 years) from first exposure.

*The Index of Dose* — The conventional index of dose is exposure, and the general method of quantifying the dose of asbestos that a person has received is by a measure of cumulative exposure. In many studies dose and exposure are used interchangeably. In fact, the concentration of asbestos to which a person is exposed is not necessarily the same as the dose of asbestos that person actually inhales.<sup>97</sup> And while ideally we may wish to know the dose inhaled, what we actually measure is exposure. In this Report, while we too use the terms interchangeably, we do so in light of the above-stated qualification. The measure of cumulative exposure is arrived at simply by multiplying the concentration of asbestos to which a worker was exposed by the number of years of exposure. However, in the case of asbestos exposure, there may be disadvantages to this general method. If the exposure is accumulated over short periods of time at high concentrations as, for example, with a demolition or maintenance worker, its effect may be different than if the same overall exposure was accumulated over a long period of time at much lower concentrations as, for example, with a worker in fixed place industry.<sup>98</sup> A measure of cumulative exposure simply does not permit exploration of these differences. For example, it does not distinguish between exposures of 2 f/cc for 50 years; 5 f/cc for 20 years; 50 f/cc for 2 years; and 100 f/cc for 1 year. All four exposures give the same measure of cumulative exposure: 100 f/cc-yrs. Further, a measure of cumulative exposure does not enable the epidemiologist to determine whether earlier exposures are more important than later exposures in the incidence of asbestos disease. While certain theoretical models have been

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<sup>96</sup>J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75"; J.C. McDonald and Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile"; Julian Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 703-711; and Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 829-836.

<sup>97</sup>Simply because a worker is exposed to a given concentration of asbestos does not necessarily mean that the individual inhales all of it.

<sup>98</sup>RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 41-42; RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, p. 54; RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 36; and RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, p. 40. See the discussion of this issue in Chapter 5, Section G of this Report.



developed to account for the residence time of asbestos in the lung,<sup>99</sup> there is no practical way to determine the real importance of residence time, and the measure of cumulative exposure used in all cohort studies treats all exposures equally, no matter how and when they were received. One final problem with asbestos studies, as with all studies of chronic disease arising from exposure to hazardous substances, is the inability to separate out completely the effects of dose from the effects of duration of exposure.<sup>100</sup> Ideally, the investigator would wish to separate completely the acquisition of exposure from the measurement of its effect so that one does not contaminate the other. The classic situation involving radiation disease where this was indeed regrettably possible was the dropping of the atomic bomb at Hiroshima and Nagasaki.<sup>101</sup> However, the follow-up period and the dose period invariably run together in asbestos cohort studies, which may blur the true nature of the dose-response relationship.

*Analysis* — The methods of analysis in the studies we have considered are sophisticated and complex and a detailed treatment in the text of this Report is not essential to understanding the significance of the results. We recognize that the analytical methods employed in the various studies differ to some extent.<sup>102</sup> However, we are satisfied that these different methods do not invalidate the results. Where appropriate, we will comment when the choice of a particular method of analysis may have affected the interpretation of the results.

### D.3 The Case-Control Study

The basic design of a case-control study is to define a group of individuals who have some specified disease (the cases) and a second group

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<sup>99</sup>Geoffrey Berry and Hilton C. Lewinsohn, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part I. Morbidity," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 185-194. Mr. Berry, in his evidence before this Commission, suggested that the traditional way of measuring dose by cumulative exposure appears unrealistic because it does not take account of when the exposure took place. The various models developed in the paper that he co-authored with Dr. Lewinsohn weighted the exposures by the time that the asbestos dust remained in the lung, but assumed that there was elimination of the dust. Thus, exposure that took place, for example, 25 years ago was considered more important in terms of disease production under the Berry and Lewinsohn models than exposure which took place, say, 5 years ago. See RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, pp. 25-27, 65-66. Dr. Crump, in his evidence, suggested that the health effect of different time patterns of exposure is not understood very well at all. See RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, pp. 22-24.

<sup>100</sup>RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 38-40; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 31-32.

<sup>101</sup>RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 31-32.

<sup>102</sup>For example, see Chapter 5, Section B of this Report.



who do not have the disease (the controls). The cases and the controls are then compared in terms of a factor or factors of interest.<sup>103</sup> One particular type of study that commonly uses a case-control analysis is a study which examines the fibre content of lung tissue in mesothelioma victims in order to assess which fibre type was responsible for causing the tumour.<sup>104</sup>

The case-control study is not considered to be as strong an analytical tool as the cohort study because it is more prone to selection bias in the control group.<sup>105</sup> Still, such analysis does provide useful evidence, particularly when it is done in conjunction with cohort analysis.

## D.4 The Case-Series Study

Here an investigator simply reports a series of cases among a defined population group. No comparison group is provided. Dr. Sackett has suggested that case series are prone to over-interpretation and are best used to stimulate further investigation of the problem by other methods.<sup>106</sup> A very good illustration of the value of a case-series report is that by Wagner, Sleggs, and Marchand of mesothelioma victims in South Africa beginning in 1960.<sup>107</sup> This case-series report not only alerted the medical community to the association between asbestos and mesothelioma, but of course stimulated the investigation of that association elsewhere in the world and by other more rigorous methods.

## E. The Human Experience: Lung Tissue Studies

Lung tissue studies concerning asbestos are of recent vintage, with most if not all documented work having been published in the last

<sup>103</sup>Sackett, "A Review of Diagnostic Tests for Causation," p. 12 and Appendix B, p. 3.

<sup>104</sup>See, for example, J.S.P. Jones et al., "The Pathology and Mineral Content of Lungs in Cases of Mesothelioma in the United Kingdom in 1976," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 187-199; Alison D. McDonald, J. Corbett McDonald, and Fred D. Pooley, "Mineral Fibre Content of Lung in Mesothelial Tumours in North America," *Annals of Occupational Hygiene* 26:1-4 (1982): 417-422; and Neil Rowlands, Graham W. Gibbs, and Alison D. McDonald, "Asbestos Fibres in the Lungs of Chrysotile Miners and Millers — A Preliminary Report," *Annals of Occupational Hygiene* 26:1-4 (1982): 411-415.

<sup>105</sup>Sackett, "A Review of Diagnostic Tests for Causation," p. 12 and Appendix B, p. 3.

<sup>106</sup>*Ibid.*

<sup>107</sup>J.C. Wagner, C.A. Sleggs, and P. Marchand, "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," *British Journal of Industrial Medicine* 17 (1960): 260-271.

decade.<sup>108</sup> They are thought to provide important evidence on two questions: first, as to whether the victim had been exposed to asbestos; and second, as to the relative pathogenicity of asbestos fibres of different types and sizes.

The typical lung tissue study is a case-control analysis in which the asbestos fibre content of the lung tissue of the series of cases (persons who died of asbestos-related disease, usually mesothelioma) is compared with a matched series of controls, with the specimen analysis done by electron microscopy. The investigator seeks to draw conclusions as to the causal relationship between exposure and disease based on whether or not the fibre content of the lungs of the cases is or is not significantly elevated above that of the controls.

The principal reservation concerning lung tissue studies is the extent to which the tissue examined at death reflects actual exposure in life. When an examiner looks at autopsy material, he is looking at what is residual and not necessarily at what was originally deposited there and may have been removed during life. This creates the concern that the fibres which may have been there initially and caused the trouble have been removed before the lung tissue is examined.<sup>109</sup> The amount of asbestos fibre retained in the lung is likely underestimated in any event due to the difficulty encountered in its recovery from the lungs at autopsy, and this difficulty likely applies more to chrysotile than to the other fibres because of chrysotile's greater solubility and its apparent tendency to break down chemically and physically after a long residence time.<sup>110</sup>

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<sup>108</sup>Jones et al., "The Pathology and Mineral Content of Lungs in Cases of Mesothelioma in the United Kingdom in 1976"; A.D. McDonald, J.C. McDonald, and Pooley, "Mineral Fibre Content of Lung in Mesothelial Tumours in North America"; and Rowlands, Gibbs, and A.D. McDonald, "Asbestos Fibres in the Lungs of Chrysotile Miners and Millers — A Preliminary Report." See also, Fred D. Pooley, "Methods for Assessing Asbestos Fibres and Asbestos Bodies in Tissue by Electron Microscopy," in *Biological Effects of Asbestos*, eds. P. Bogovski et al., IARC Scientific Publications, no. 8 (Lyon, France: International Agency for Research on Cancer, 1973), pp. 50–53.

<sup>109</sup>RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 100; and RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 90.

<sup>110</sup>Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 194.

## F. Animal Studies: The Advantages and Disadvantages of Animal Experiments with Asbestos

Animal experiments have become an important aspect of the estimation of the risk to humans of cancer and other diseases;<sup>111</sup> and animal testing has been widely used to study the health effects of asbestos. Three different types of animal experiments are relevant for the study of asbestos-related disease: injection experiments, inhalation experiments, and ingestion experiments.

The underlying premise of animal experiments is that the effects of properly conducted animal tests are applicable to humans. Considerable assurance as to the general applicability of this premise is derived from the fact that compounds generally known to be carcinogenic in humans are also carcinogenic in animals (save arsenic and perhaps benzene) and from the fact that no demonstrably significant aspect of the natural occurrence, induction, and properties of cancer has been shown to differ fundamentally between humans and experimental animals.<sup>112</sup>

Having regard to these considerations, we accept the conventional wisdom of the medical and scientific community that animal experiments are useful as a qualitative indication of potential human health effects in relation to asbestos exposure. Their great advantage is that they can be done over shorter time with greater precision and control than can be achieved by epidemiological methods.<sup>113</sup> Animal studies enable the investigator to explore particular issues for which the data from human experience may not be available or may not be reliable. For example, they permit an investigation of the importance and effect of variations in fibre size, the break-up of fibres in lung tissues, and the effects of different fibre types under controlled conditions.

While we recognize the value and importance of the results of animal testing with asbestos, we are also mindful of their limitations. One such limitation is that the asbestos samples generally used in animal experiments to date have not been used commercially. For example, neither the superfine Canadian chrysotile nor the standard UICC (Union Internationale

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<sup>111</sup> Crump et al., "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," p. 2973; and "The OSHA Cancer Policy," pp. 5060ff.

<sup>112</sup> "The OSHA Cancer Policy," pp. 5060-5067; and John M.G. Davis, "The Use of Animal Models for Studies on Asbestos Bioeffects," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 795-798.

<sup>113</sup> Ibid. RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 39-40, 143; RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, p. 60; and RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, p. 11.

Contre le Cancer) chrysotile, amosite, or crocidolite which are utilized in much of the experimental work are used in industry.<sup>114</sup> The fibres in these experimental samples are indeed usually much finer than their industrial counterparts, and in the result their use in animal experiments blurs the potential differences between the effects of fibre type and of fibre dimension. Of course, to the extent that the trend in modern asbestos industry is to utilize finer fibres, animal experiments using UICC samples become that much more relevant, a point that was made by Dr. E. Donald Acheson in his report to the U.K. Advisory Committee on Asbestos and in his evidence before us.<sup>115</sup>

The most precise of animal experiments, the intrapleural injection experiment, is also the most unrealistic in comparison with the human experience.<sup>116</sup> Humans breathe or ingest asbestos fibres. Injection experiments implant or inject dust containing asbestos onto the lung or pleura, thereby avoiding the need to travel to the site of interest and bypassing the defence mechanisms of the respiratory system. However unrealistic they may be in terms of how the fibres reach the pleura, intrapleural injection experiments still do allow the investigator to test directly the effects of asbestos once deposited in the pleural cavity. Inhalation experiments are more realistic than injection experiments in that they more closely simulate human exposure, but they are also harder to interpret since the penetration of dust through the airways and alveoli will vary with time and will differ with different samples of dust. For the results to be of value, the dust cloud must be uniform and accurately monitored. However, Dr. Davis testified that in most animal inhalation experiments, there is a relatively poor characterization of the dust cloud in terms of two possibly important variables: fibre size and fibre shape.<sup>117</sup>

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<sup>114</sup>RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 29, 76-77; John M.G. Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature* (Edinburgh: Institute of Occupational Medicine, December 1980), pp. 17-19; and RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 33.

<sup>115</sup>E. Donald Acheson and Martin J. Gardner, "The Ill Effects of Asbestos on Health," in U.K., Advisory Committee on Asbestos, *Asbestos — Volume 2: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979), paragraph 168, pp. 33-34; and RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 11. See also, J. Christopher Wagner et al., "The Effects of the Inhalation of Asbestos in Rats," *British Journal of Cancer* 29:3 (March 1974): 268.

<sup>116</sup>J. Christopher Wagner, Geoffrey Berry, and Victor Timbrell, "Mesotheliomata in Rats After Inoculation with Asbestos and Other Materials," *British Journal of Cancer* 28:2 (August 1973): 177; and Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, p. 11.

<sup>117</sup>Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, p. 11.



A frequent criticism of animal experimental studies is that due to inter-species differences, extrapolation to humans is not justified. While this may be an extreme view, it is important that the animal model for experimental pathologic studies should duplicate as closely as possible the known human pathologic conditions intended to be studied.<sup>118</sup> Most experimental animal studies of asbestos disease have used the rat because the biological mechanisms of importance as between the rat and the human are thought to be similar.<sup>119</sup> However, in at least one respect, the tissue reaction of rats to asbestos fibres is dissimilar to that of humans. Rats rarely, if ever, form asbestos bodies. At present, it is uncertain whether the inability of rats to form these bodies is a disadvantage to their use in animal experiments.<sup>120</sup>

In order to induce cancer in animals within their lifetime, the doses of asbestos to which animals are exposed in laboratory testing are extremely high, certainly much higher than those to which workers are likely to have been exposed in recent years.<sup>121</sup> This gives rise to another important limitation on the value of animal experiments with asbestos. They cannot at the present time give precise information on the quantitative risk of exposure in humans.<sup>122</sup> Accordingly, while animal experiments are useful in a qualitative sense to indicate the existence of a dose-response relationship, they are of dubious assistance in determining the shape of the dose-response curve in humans. Because animals are given high doses over short periods, quantitative comparison with, or quantitative extrapolation to, the human experience is of doubtful reliability.

Laboratory animals have a much shorter lifespan and therefore a much shorter period for tumour induction than humans. This shorter time period may mean that any potential health effects in humans arising from the residence time of fibres in the lungs or arising from the physical process by which fibres break up may not be discoverable from animal experiments. More generally, animal experiments with asbestos will not be able to cast any light on the long-term chemical effects of fibres if indeed such effects

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<sup>118</sup>Davis, "The Use of Animal Models for Studies on Asbestos Bioeffects," pp. 795-796.

<sup>119</sup>Ibid. See also, Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, pp. 17-19; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 134-135. Dr. Davis testified that the size of the air space of the alveoli is not too much different in the rat than in the human. He also indicated that the macrophages for a rat are very similar in size as for a human and that the rat will be inhaling from any dust cloud probably the same fraction of asbestos fibres compared with body size and weight as the human being.

<sup>120</sup>Davis, "The Use of Animal Models for Studies on Asbestos Bioeffects," p. 796.

<sup>121</sup>Crump et al., "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," p. 2973; and "The OSHA Cancer Policy," pp. 5060ff.

<sup>122</sup>RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 9.

exist.<sup>123</sup> While the rat's shorter lifespan may in one sense detract from the value of animal experiments, it is very advantageous from another point of view. It allows identification of the various effects of a substance well before the results of epidemiological studies in humans are known. This makes animal experiments particularly important in assessing the disease potential of new substances or substitutes for currently used substances.<sup>124</sup> But even with a substance such as asbestos where there is already a body of epidemiological data, animal experiments may provide new information as to its health effects and, where appropriate, enable preventive action to be taken in advance of such information being made known from epidemiology.

## G. *In Vitro* Studies

*In vitro* or cell culture studies have been used as an alternative to animal experiments to investigate the biologically relevant characteristics of asbestos. For example, several authors have demonstrated the dependence of toxicity on fibre length in *in vitro* systems. Others have demonstrated that durability or solubility of fibres may be an important factor and that chrysotile in particular can be broken down in organisms.<sup>125</sup>

The advantages of *in vitro* experiments, in addition to the considerable savings in time and cost that they occasion, are that they provide an opportunity to study events at the cellular level under controlled, quantifiable, and reproducible conditions. Their disadvantage is that they take place in isolation from the normal integrating mechanisms to which the cell is subjected in the whole organism. The extrapolation of the results of *in vitro* studies with asbestos to humans or even animals has been limited, and it would appear that they are best used to supplement the findings of epidemiology and animal experiments in relation to the pathogenesis of asbestos.<sup>126</sup>

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<sup>123</sup>RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 46; RCA Transcript, Evidence of Mr. Julian Peto, 30 July 1981, Volume no. 25(B), p. 136; Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, pp. 17-19; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 30-32. If the time for break-up of chrysotile is roughly the same in animals as in humans, then it would take place over most of the rat's lifetime, but a very short percentage of a human's lifetime.

<sup>124</sup>"The OSHA Cancer Policy," pp. 5060ff.; and Crump et al., "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," p. 2973.

<sup>125</sup>E.G. Beck, "Experimental Pathology — *In Vitro* Studies — Related to Asbestos and Other Mineral Fibres," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 385-400.

<sup>126</sup>*Ibid.*, p. 385.



## **Chapter 5    The State of the Evidence on Major Health Questions**

### **A. Introduction**

In Sections B, C, and D of this chapter we address the question: What are the important determinants of the health hazards of asbestos? First we consider the use of asbestos in different types of industrial processes. We do so by reviewing the major cohort studies of asbestos workers. The findings of these studies appear to indicate that the health hazards of asbestos vary significantly between different industrial processes. We then consider the health effects of different fibre types. Here the epidemiological data appear to indicate that the amphiboles are more hazardous than chrysotile, particularly in relation to the production of mesothelioma. We then discuss what might account for these differences. The available data indicate that there is an association between fibre dimension and asbestos-related disease. Long, thin fibres, that is, fibres longer than 5 or perhaps 8 microns in length and thinner than 1.5 or perhaps 0.25 microns in diameter, appear to be particularly implicated in the pathogenicity of asbestos. And just as there is an association between fibre dimension and disease, there appears in turn to be an association between type of industrial process and fibre dimension. As well, there appears to be a certain association between fibre type and fibre dimension.

While we consider that the dimension of the fibres offers the main explanation for the health hazards arising from asbestos, we acknowledge that other factors — fibre chemistry and durability — may play a role. We also acknowledge that our conclusions must be couched in a degree of uncertainty. Just as there exists medical uncertainty as to the mechanisms by which the asbestos-related diseases are caused, so there remains uncertainty as to the biologically relevant characteristics of asbestos.



In Sections E, F, G, H, and I of this chapter we consider some specific health issues with policy implications. In Section E we examine the nature of the dose-response relationship for the asbestos-related diseases and conclude that a linear relationship satisfactorily describes the dose-response relationship for asbestosis, lung cancer, and mesothelioma. We also find that while the weight of the available data indicates that there is now a low level of occupational exposure at or below which the clinical manifestation of asbestosis is most unlikely, we find that no such conclusion is justified either for lung cancer or mesothelioma.

In Section F we consider the effect of smoking on the incidence of disease. We note that smoking cannot initiate either mesothelioma or asbestosis, although it may enhance the progression of the latter. In contrast, not only can smoking initiate lung cancer, but in combination smoking and asbestos appear to act synergistically in the production of the tumour.

In Section G we consider the health effects of peak exposures or short, intense doses of asbestos. We do so out of concern that exposure of this kind is characteristic of occupations that bring workers into contact with asbestos through demolition, building maintenance, and repair work. From the limited data available we conclude that short, peak exposures to asbestos may pose a special health risk because of the possibility that such exposures will overwhelm the lung's clearance mechanisms.

We then discuss in Section H whether there is a special health risk to children from asbestos exposure. While we do not suggest that the levels of asbestos exposure outside the workplace themselves present a health risk to children or to any other members of the general public, we do indicate that the time course of asbestos-related disease, particularly mesothelioma, means that the earlier in life asbestos exposure first takes place, the greater the risk of disease.

Finally, we consider in Section I whether there is any health risk from the ingestion of asbestos fibres either through eating or drinking. This issue has raised public concern especially because of the presence of asbestos in many municipal water systems. We find that it is most unlikely that ingested fibres penetrate the gastrointestinal tract. In addition, there is a great deal of evidence that asbestos fibres found in water systems are very short and hence are not to be considered hazardous. (See Chapter 11 of this Report.) We conclude that the evidence fails to indicate any health risk from the ingestion of asbestos fibres.

## B. The Effect of Industrial Process

### B.1 Asbestos Mining

#### (a) Quebec

In 1966, a research team at McGill University headed by Dr. J. Corbett McDonald began investigating the health effects of a large group of chrysotile miners and millers working in the Eastern Townships of Quebec.<sup>1</sup> Dr. J.C. McDonald and his colleagues have reported the results of their findings several times over the years and have continued to update their data and improve their methodology.<sup>2</sup> The most recent report of their findings was published in 1980 and was based upon a cohort of 11,379 workers (10,939 men and 440 women) born between 1891 and 1920 who had worked for at least one month in the mines and mills of Asbestos and Thetford Mines, Quebec, and who had been followed to the end of 1975.<sup>3</sup> It is generally conceded that this report represents the most comprehensive and reliable epidemiological study of the mining and milling of chrysotile asbestos.<sup>4</sup> There is considerable exposure information. The authors identified 5,783 different jobs through detailed work histories. Individual

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<sup>1</sup>Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 10.

<sup>2</sup>J. Corbett McDonald et al., "Mortality in the Chrysotile Asbestos Mines and Mills of Quebec," *Archives of Environmental Health* 22 (June 1971): 677-686; J. Corbett McDonald et al., "The Health of Chrysotile Asbestos Mine and Mill Workers of Quebec," *Archives of Environmental Health* 28:2 (February 1974): 61-68; J. Corbett McDonald and F. Douglas K. Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 1-9; and J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," *British Journal of Industrial Medicine* 37 (1980): 11-24. Dr. J.C. McDonald and his colleagues have studied the Quebec miners and millers at four points in time: 1966, 1969, 1973, and 1975. See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 10. For a detailed discussion of the methods of cohort analysis used by Dr. J.C. McDonald, see F.D.K. Liddell, J.C. McDonald, and D.C. Thomas, "Methods of Cohort Analysis: Appraisal by Application to Asbestos Mining," *Journal of the Royal Statistical Society* 140, Part 4 (1977): 469-491.

<sup>3</sup>J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75." In this latest report, Dr. J.C. McDonald and his colleagues used two main methods of analysis: a man-years approach (*a priori* analysis) and a case-controls approach (*a posteriori* analysis). RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 6.

<sup>4</sup>Michael Alavanja, Christine New, and Judy Parsells, "An Assessment of the Epidemiological Literature Related to Worker Exposure to Chrysotile in Mines and Mills," NIOSH internal document prepared for the U.S. Mine Safety and Health Administration, 1981. (Mimeographed.); RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 103; RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, p. 85; and RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 62.

cumulative dust estimates were made on the basis of over 4,000 midjet impinger dust counts made personally by the same industrial hygienist in the years 1949–1966.<sup>5</sup> However, it must be observed that there were no measurements made in the open pit, only a limited number of measurements in the underground mines, and virtually no actual measurements of any kind in the pre-1949 period.<sup>6</sup> For the period prior to 1949, estimates had to be made on the basis of comparisons with more recent conditions following interviews with long-service employees.<sup>7</sup> Moreover, those measurements made after 1949 were of dust particles rather than asbestos fibres. While Dr. J.C. McDonald and his associates have recently been attempting to convert the dust particle measurements to fibre measurements,

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<sup>5</sup> J.C. McDonald et al., “Dust Exposure and Mortality in Chrysotile Mining, 1910–75,” p. 13. Dr. Graham W. Gibbs testified that he collected the data which Dr. Maurice Lachance had available for the mining industry for each year and by work area. He then looked at the concentrations and the pattern of dustiness for each work area over a period of time. Dr. Gibbs then took each of the jobs that each of the people in the industry had and looked at which work areas those jobs would have been associated with. He then assigned, for those years to those jobs, the dust concentrations associated with those particular work areas. See RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 29–30.

<sup>6</sup> J.C. McDonald et al., “Dust Exposure and Mortality in Chrysotile Mining, 1910–75,” p. 13; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 5; and RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, p. 77.

<sup>7</sup> RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 29–31. Dr. Gibbs testified that the general view of long-term employees and of management was that dust conditions in the early 1950s reflected fairly well dust concentrations that existed almost back at the turn of the century. Accordingly, Dr. Gibbs and Dr. Lachance utilized the concentrations in the early 1950s to extrapolate backwards, modified somewhat by the information available from the employee and management groups. Dr. Gibbs is of the view that the raw dust data from the Quebec mines are probably far better than the data found for most epidemiological studies in the sense that although there are not measurements for every day and every year at all locations, there are essentially systematic measurements made by the same individual over a long period of time. See RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 58.

he frankly conceded in his testimony before this Commission that the results have been far from satisfactory.<sup>8</sup>

By the end of 1975, 4,463 men and 84 women (a total of 4,547 workers) had died out of the cohort of 11,379 workers (10,939 men and 440 women). There was a net excess of 33.9 deaths at Asbestos and 208.8 at Thetford Mines, yielding overall standard mortality ratios (SMRs) of 102 and 110 respectively. Approximately 10% of the cohort was not traced, but most were apparently workers with short service.<sup>9</sup>

The data, analyzed by different methods, gave results consistent with one another and with previous analysis. For total mortality, and for mortality from pneumoconiosis (which would be mostly asbestosis) and lung cancer, there were quite consistent trends to higher SMRs the greater the dust exposure.<sup>10</sup> The pattern for gastrointestinal cancer was uneven: there was substantial excess mortality from cancer of the upper gastrointestinal tract in the workers most heavily exposed (at Thetford Mines), but otherwise no excess, prompting the authors to suggest that some other causal factor was operative.<sup>11</sup>

There are at least two noteworthy findings of this study. First, among the 1,904 men with at least 20 years' employment in the lower dust categories, with an average yearly exposure of 6.6 million particles per

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<sup>8</sup> J. Corbett McDonald, Graham W. Gibbs, and F. Douglas K. Liddell, "Chrysotile Fibre Concentration and Lung Cancer Mortality: A Preliminary Report," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 811-817. The authors used a case-control analysis of 244 deaths from lung cancer and 244 surviving controls matched for date of birth, mining area, and smoking habit. All measurements ever made in the industry using membrane filters or midget impingers were compiled although there was little overlap. These measurements yielded an average conversion ratio of 1 mppcf equals 3.14 f/cc. In the article, the authors suggested that, in the absence of evidence to the contrary, this average conversion ratio could usefully be applied to other results in the chrysotile mining industry. However, in his oral testimony before this Commission, Dr. Gibbs indicated that he was unhappy with this conclusion and would have preferred to have restricted the conversion factor of 3.14 as applicable only to the precise job distribution in the case-control study from which the figure came. See RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 66. In his own testimony, Dr. J.C. McDonald conceded that his group's attempt to convert dust particle measurements to fibre measurements is "... more or less fizzling out, actually. I mean, we're beginning to get depressed about it. We've been doing it for a good five or six years, and I think we know how almost unanswerable the problem is." See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 42.

<sup>9</sup> J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," p. 11. The authors' mortality analyses focused on the 3,291 male deaths occurring 20 or more years from first employment and in the period 1951-1975. The figure 3,291 is used in both Tables 5.1 and 5.18 in this chapter.

<sup>10</sup> Ibid., p. 17.

<sup>11</sup> Ibid., pp. 22-23.



cubic foot (mppcf) [or perhaps about 20 fibres per cubic centimetre (f/cc)], excess mortality, although not absent, was not considered to be statistically significant except for asbestosis.<sup>12</sup> The inability of such a large epidemiological study to demonstrate an increased risk for cancer at exposure levels far in excess of present levels suggests that any risk in chrysotile mining at such levels is very small. Indeed, the authors speculated in their published study that the dust concentrations in the Quebec mines and mills in the early 1950s carried a lung cancer risk equivalent to heavy smoking, whereas at more recent concentrations of around 1 mppcf the order of risk may approximate to less than one cigarette per day.<sup>13</sup> It may be that the cancer risk has been underestimated to some extent in this cohort, in part because the authors used provincial mortality data for their comparisons whereas the rural mining area of Quebec has a lung cancer mortality rate which is two-thirds of the provincial rate.<sup>14</sup> Still, it is evident that the overall disease incidence among these chrysotile miners and millers is relatively low save in the long-term and most heavily exposed employees.

The second important finding of this study was that among the 4,547 deaths in this cohort, there were only 11 mesothelioma deaths, all of them pleural, 10 men and one woman.<sup>15</sup> Subsequent investigation by Dr. Alison D. McDonald and consideration of cases among those who did not qualify for inclusion in the cohort have increased the number to 21, 5 of whom also worked at a nearby factory which used both crocidolite and chrysotile.<sup>16</sup> Table 5.1 shows the standard mortality ratios for various causes of death in relation to duration of service for Dr. J.C. McDonald's cohort.

A smaller cohort of the Quebec miners has also been studied by Dr. William J. Nicholson and his colleagues at the Mount Sinai School of Medicine in New York.<sup>17</sup> They examined all men employed during 1961 at Thetford Mines with at least 20 years' seniority. The total cohort thus consisted of 544 persons, of whom 178 were dead by the end of 1977. The

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<sup>12</sup> Ibid., pp. 11, 23. In his evidence, Dr. J.C. McDonald emphasized that this finding did not indicate that there is no risk at such exposure. Rather, his conclusion was that there is a risk but that it cannot be demonstrated with statistical significance. See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 23.

<sup>13</sup> J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," p. 21.

<sup>14</sup> RCA Transcript, Evidence of Dr. William J. Nicholson, 30 June 1981, Volume no. 15, p. 40; and Alavanja, New, and Parsells, "An Assessment of the Epidemiological Literature Related to Worker Exposure to Chrysotile in Mines and Mills," p. 12.

<sup>15</sup> J.C. McDonald and Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile," pp. 6-7; and J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," p. 11.

<sup>16</sup> Alison D. McDonald, "Malignant Mesothelioma in Quebec," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 673-680.

<sup>17</sup> William J. Nicholson et al., "Long-Term Mortality Experience of Chrysotile Miners and Millers in Thetford Mines, Quebec," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 11-21.

Table 5.1\*  
Deaths, by Cause, in Relation to Duration of Service

Cause of Death	Length of Gross Service (Years)									
	Very Short (<1) O** SMR***	Short (1<5) O SMR	Medium (5<20)		Long (≥20)		Complete Cohort			
			O	SMR	O	SMR	O	SMR	O	SMR
All causes	885	1.07	629	1.09	679	1.15	1,098	1.07	3,291	1.09
Pneumoconiosis	1	1.15	3	5.00	2	3.39	36	34.62	42	13.55
Malignant neoplasms										
Lung	47	0.97	29	0.83	50	1.37	104	1.61	230	1.25
Esophagus and stomach	37	1.30	25	1.27	18	0.91	50	1.47	130	1.27
Colon and rectum	22	0.78	13	0.67	23	1.16	21	0.62	79	0.78
Other abdominal	20	1.08	12	0.92	14	1.04	21	0.90	67	0.98
Larynx	6	1.48	5	1.75	1	0.34	4	0.78	16	1.07
Other	67	1.12	43	1.04	48	1.13	79	1.08	237	1.09
Heart disease	370	1.06	251	1.02	287	1.15	424	0.97	1,332	1.04
Respiratory tuberculosis	7	0.62	7	0.89	21	2.68	22	1.56	57	1.39
Other respiratory	29	0.66	46	1.52	22	0.71	59	1.12	156	0.99
Cerebrovascular	62	0.95	49	1.12	50	1.13	82	1.11	243	1.07
Accidents	52	1.36	38	1.32	37	1.18	56	0.96	183	1.17
All other known causes	130	1.03	94	1.07	94	1.05	132	0.85	450	0.98
Cause not known	35	—	14	—	12	—	8	—	69	—

**Notes:** \* [For all tables in this chapter, "O" or "Obs" mean observed deaths; "E" or "Exp" mean expected deaths; and "SMR" means standard mortality ratio.]

\*\* Columns headed O give the numbers of deaths of men, 20 years or more after first employment, occurring during 1951-1975.

\*\*\* Figures under headings SMR are ratios of deaths observed to those expected on the basis of male mortality in Quebec.

SOURCE: J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," *British Journal of Industrial Medicine* 37 (1980): 17 (Table 6).

SMRs for deaths from all causes and for lung cancer were 111 and 252 respectively. There was only one mesothelioma in the cohort and no excess of gastrointestinal cancer.<sup>18</sup> The cohort studied by Dr. Nicholson was heavily exposed and his results appear to be quite compatible with those of Dr. J.C. McDonald's most heavily exposed group for which the SMRs for death from all causes and for lung cancer were 130 and 225 respectively.<sup>19</sup>

### (b) Italy

The disease incidence in chrysotile mining has also been investigated by Rubino et al. who examined the health experience of a group of chrysotile miners at the Balangero Mine in Northern Italy.<sup>20</sup> The cohort studied was quite small, consisting of 952 miners who had worked at least one month between 1930 and 1965 (and who were known to be alive in 1946). There were 332 deaths, giving an SMR for deaths from all causes of 155. Significant increases over expected were found, *inter alia*, for laryngeal cancer and non-malignant respiratory diseases, but not for lung cancer, where there were only 11 deaths and an SMR of 106. There was only one death from mesothelioma (pleural) which was included in the lung cancer calculation. However, the risk of asbestos-related disease may be underestimated in this study since mortality was examined as of the end of 1975, leaving a latency period of less than 20 years for individuals initially employed after 1955.<sup>21</sup> Dr. J.C. McDonald noted that the findings among the Italian miners were very similar to his own.<sup>22</sup>

### (c) Australia

There has been at least one cohort study of crocidolite mining, that by Hobbs et al. who recently investigated a group of Western Australian

<sup>18</sup>Ibid., pp. 11–16. In 130 cases, the authors used clinical, surgical, or pathological data to supplement the information on the death certificates as to cause of death.

<sup>19</sup>See J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910–75," Table 8, p. 19; and RCA Transcript, Evidence of Dr. William J. Nicholson, 30 June 1981, Volume no. 15, pp. 75–77. It is to be noted that Dr. Nicholson et al. studied only employees at Thetford Mines whereas Dr. J.C. McDonald et al. studied employees at both Asbestos and Thetford Mines. Overall, those employed at Asbestos had a lower mortality and disease experience than those employed at Thetford Mines.

<sup>20</sup>G.F. Rubino et al., "Mortality of Chrysotile Asbestos Workers at the Balangero Mine, Northern Italy," *British Journal of Industrial Medicine* 36 (1979): 187–194. The authors noted that the death certified as being caused by pleural mesothelioma occurred 35 years after starting employment in a worker with 33 years' exposure. Since there was no histological confirmation of the diagnosis, the authors accepted the diagnosis only with reservation.

<sup>21</sup>Alavanja, New, and Parsells, "An Assessment of the Epidemiological Literature Related to Worker Exposure to Chrysotile in Mines and Mills."

<sup>22</sup>J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910–75," p. 22.

crocidolite miners.<sup>23</sup> The study was based upon 6,200 male employees of the principal mining company in the region, followed to the end of 1977. No minimum interval from first employment was defined for this study, and accordingly, for many in the cohort the follow-up period was quite short. The SMR for death from all causes was only 90, perhaps in part attributable to a healthy worker effect. (See Chapter 4, Section D.2 of this Report.) However, the SMR for lung cancer was 157 (60 deaths versus 38.24 expected) and for pneumoconiosis, 1,045 (14 against 1.34 expected).<sup>24</sup> There were 26 cases of mesothelioma, all pleural, with a median interval from first exposure of approximately 20 years.<sup>25</sup> The incidence of mesothelioma was strongly related both to the duration and intensity of the crocidolite exposure.

#### (d) South Africa

Within South Africa, crocidolite, amosite, and chrysotile have all been mined for many years. According to the final report of the United Kingdom Advisory Committee on Asbestos (the Simpson Report), the crocidolite mined in South Africa is of two types: one with extremely fine fibres which is mined in the northern part of the Cape province and the other, a more coarse variety mined in the Transvaal.<sup>26</sup> The amosite mines are found close to the crocidolite mines in the Transvaal, and the chrysotile mines are found in the south-east near the Swaziland border.

<sup>23</sup>M.S.T. Hobbs et al., "The Incidence of Pneumoconiosis, Mesothelioma and Other Respiratory Cancer in Men Engaged in Mining and Milling Crocidolite in Western Australia," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 615-625.

<sup>24</sup>*Ibid.*, p. 621. The authors have noted that the mortality ratio for all causes of death in the cohort, excluding both respiratory disease and accidents, is 0.80. Thus, there is a twofold difference between the mortality ratio for respiratory cancer and those for other causes of death.

<sup>25</sup>*Ibid.*, pp. 618-620. The 26 cases of mesothelioma included cases diagnosed to July 1, 1979. The authors' mortality study, which indicated 17 mesothelioma deaths, was restricted to deaths before January 1, 1978. Mr. Julian Peto has noted the absence of peritoneal mesothelioma among the Western Australian crocidolite miners, which is in contrast to the other data which suggest that the risk of peritoneal mesothelioma is substantial after crocidolite exposure. See Julian Peto, "Discussion Summary," in *Biological Effects of Mineral Fibres*, vol. 2, p. 731. In their update on "The Ill Effects of Asbestos on Health" (which was prepared for the U.K. Advisory Committee on Asbestos), Dr. E. Donald Acheson and Dr. Martin J. Gardner have noted, on the basis of a personal communication from M.S.T. Hobbs, that as of 1983 there were 64 cases of mesothelioma among the Western Australian crocidolite miners: 60 pleural and 4 peritoneal. See E. Donald Acheson and Martin J. Gardner, *Asbestos: The Control Limit for Asbestos*, prepared for the U.K. Health and Safety Commission (London: Her Majesty's Stationery Office, 1983), paragraph 24, p. 4.

<sup>26</sup>E. Donald Acheson and Martin J. Gardner, "The Ill Effects of Asbestos on Health," in U.K., Advisory Committee on Asbestos, *Asbestos — Volume 2: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979), paragraph 130, p. 28.



Despite the extensive asbestos mining that has taken place in South Africa, there has been no systematic epidemiological study of the workers at risk so that case reports of disease must be considered cautiously. Nonetheless, it is noteworthy that according to Professor Ian Webster, of the 235 cases of mesothelioma reported in South African miners between 1956 and 1982, almost all were exposed to Cape crocidolite, a few to Transvaal amosite, and an occasional one to Transvaal crocidolite. No cases of mesothelioma were reported in relation to the mining of chrysotile but the chrysotile mining operation in South Africa is relatively small.<sup>27</sup>

### (e) Asbestos-Contaminated Mines

In addition to asbestos deposits, asbestiform fibre concentrations are often found in other mineral deposits. For example, Mr. Richard A. Lemen of the United States National Institute for Occupational Safety and Health (NIOSH) testified before the Commission that data collected by the Institute indicated there were a number of mining operations in the United States in which at least measurable amounts of asbestos-like fibres were found.<sup>28</sup> Two examples were the Homestake Gold Mine located at Lead, South Dakota, where miners were occupationally exposed to fibrous cummingtonite-grunerite, that is, amosite; and the talc mines in New York State which are contaminated with deposits of tremolite.

The gold mine in South Dakota has now been subjected to three separate cohort studies.<sup>29</sup> An initial investigation by Gillam et al., published by NIOSH in 1976, focused on those 440 workers who by 1960 had completed at least 5 years of underground mining and found that there was excess mortality due to malignant and non-malignant respiratory disease particularly in the period 20 or more years from first exposure. The authors of the study concluded that neither silica exposure nor cigarette smoking could account for the observed increased lung cancer risk among these hard rock gold miners and accordingly attributed it to the exposure to the

<sup>27</sup> Letter from Professor Ian Webster, National Centre for Occupational Health, Department of Health, Welfare and Pensions, Republic of South Africa to the Royal Commission on Asbestos, 14 July 1983. The findings of the Asbestos Tumour Reference Panel in South Africa list a total of 1,120 cases of mesothelioma between the years 1956 and 1982. Of this figure, 235 cases were attributed to exposure in mining and 178 cases to environmental exposure. In 453 of the cases, the source of exposure was either unknown or not available.

<sup>28</sup> RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, p. 63.

<sup>29</sup> J. Dean Gillam et al., "Mortality Patterns Among Hard Rock Gold Miners Exposed to an Asbestiform Mineral" (NIOSH Study), *Annals of the New York Academy of Sciences* 271 (28 May 1976): 336-344; J. Corbett McDonald et al., "Mortality After Long Exposure to Cumingtonite-Grunerite," *American Review of Respiratory Disease* 118:2 (August 1978): 271-277; and Samuel D. Kaplan and William R. Gaffey, "Miners Exposed to Amphibole Mineral: A Retrospective Cohort Mortality Study" (SRI Study), Cincinnati, Ohio, U.S. Department of Health and Human Services, June 1981. (Mimeographed.)

cumingtonite-grunerite.<sup>30</sup> Yet, a subsequent investigation of the same mine but of a differently constituted cohort by Dr. J.C. McDonald et al. could find no excess risk from lung cancer or any other malignancy.<sup>31</sup> Dr. J.C. McDonald conducted a mortality study of 1,358 workers who by 1973 had worked at least 21 years with the mining company. Almost all had spent most of their working lives underground. There was an increase in non-malignant respiratory mortality which appeared characteristic of hard rock mining with a severe silicotic risk.

In light of the conflicting results of these two studies, NIOSH commissioned yet a further and more comprehensive study of the same mine by the Stanford Research Institute (SRI).<sup>32</sup> In this investigation, the study cohort consisted of 3,144 men employed underground in the mine for one year or more between 1940 and 1964 and followed to the end of 1977, by which time 827 men had died. The report of the SRI investigation, which was released in draft during the course of our own inquiry, strikingly confirmed the findings of Dr. J.C. McDonald and found no excess risk of lung cancer or indeed any other cause of mortality that could be associated with the grunerite exposure.<sup>33</sup> The authors of the report concluded that members of the cohort were at high risk of death from accidents and from tuberculosis and non-malignant respiratory disease related to silica exposure. The SRI authors also commented on the different results produced by the two earlier studies and observed that the study by Gillam et al. overestimated the lung cancer risk due to the lack of up-to-date mortality data for their analysis.<sup>34</sup>

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<sup>30</sup>Gillam et al., "Mortality Patterns Among Hard Rock Gold Miners Exposed to an Asbestiform Mineral," pp. 342-343.

<sup>31</sup>J.C. McDonald et al., "Mortality After Long Exposure to Cumingtonite-Grunerite," pp. 275-276. The authors of this study have suggested that the conclusions of Gillam et al. were weakened by the observation that the odds ratio for death from respiratory malignancies was greater within 20 years of first employment than after it. On the other hand, Mr. Richard A. Lemen, a co-author of the NIOSH study by Gillam et al., suggested in his testimony that Dr. J.C. McDonald and his colleagues were looking at a survivor population and, therefore, may have underestimated the risk. See RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, pp. 141-144. Dr. John M. Dement made the same point in his testimony. See RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, pp. 50-51.

<sup>32</sup>Kaplan and Gaffey, "Miners Exposed to Amphibole Mineral: A Retrospective Cohort Mortality Study."

<sup>33</sup>Ibid.

<sup>34</sup>Ibid. To calculate their expected numbers Gillam et al. used mortality rates for the general population only through the year 1967. The cohort was followed between the years 1960 and 1973, which means that for nearly one-half the person-years of follow-up, up-to-date general population mortality rates were not available and the earlier 1967 rates had to be used. The authors of the SRI study have suggested that for most causes of death this would have had little impact but that lung cancer mortality rates for males were increasing rapidly during that period of time. Thus, the expected number of deaths for lung cancer was unavoidably underestimated in the study by Gillam et al., which means that the corresponding SMR for lung cancer was unavoidably overestimated.

It is noteworthy that the cummingtonite-grunerite fibres found at this mine, while similar to amosite fibres in chemical constitution, were generally very short. Most were less than 5 microns in length with a median fibre length of 1.1 microns, a factor which, as we shall discuss, is consistent with the finding that these fibres played no role in the disease incidence at this mine.<sup>35</sup>

As in the United States, there are in Ontario several mining operations with low levels of asbestiform fibre concentrations. For example, chrysotile can be found in the talc and magnesite deposits in the Kirkland Lake - Timmins area.<sup>36</sup> The Ministry of Labour has recently taken measurements of the asbestos levels in some non-asbestos mines in Ontario and found that they are well below the current occupational control limit for chrysotile asbestos of 1 f/cc.<sup>37</sup>

## B.2 Asbestos Manufacturing — Friction Materials

### (a) Ferodo Plant, Derbyshire, England

Mr. Geoffrey Berry and Dr. Muriel L. Newhouse have recently conducted a mortality study of workers manufacturing friction materials at the Ferodo plant in Derbyshire, England. Mr. Berry reported the preliminary results of this study during his testimony before this Commission, and the authors' final report was published following the conclusion of our hearings.<sup>38</sup>

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<sup>35</sup> John M. Dement, Ralph D. Zumwalde, and Kenneth M. Wallingford, "Discussion Paper: Asbestos Fiber Exposures in a Hard Rock Gold Mine," *Annals of the New York Academy of Sciences* 271 (28 May 1976): 345-352.

<sup>36</sup> Ulrich Kretschmar and Dianne Kretschmar, *Talc, Magnesite, and Asbestos Deposits in the Kirkland Lake - Timmins Area, Districts of Timiskaming and Cochrane*, Ontario Geological Survey Open File Report 5391 (Toronto: Ontario Ministry of Natural Resources, 1982).

<sup>37</sup> Letter from Mr. Hugh M. Nelson, Special Assistant, Industrial Hygiene, Occupational Health and Safety Division, Ontario Ministry of Labour to the Royal Commission on Asbestos, 14 September 1981.

<sup>38</sup> The preliminary results were presented at the Fifth International Symposium on Inhaled Particles, conducted by the British Occupational Hygiene Society at Cardiff, Wales, in September 1980; and were subsequently published as: Muriel L. Newhouse, Geoffrey Berry, and Joseph W. Skidmore, "A Mortality Study of Workers Manufacturing Friction Materials with Chrysotile Asbestos," *Annals of Occupational Hygiene* 26:1-4 (1982): 899-909. For the final report, see Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 1-7. The dust conditions in the plant have been separately discussed in J.W. Skidmore and B.L. Dufficy, "Environmental History of a Factory Producing Friction Material," *British Journal of Industrial Medicine* 40:1 (February 1983): 8-12.



The study was based on a cohort of 13,460 workers employed from 1942 onwards and traced to the end of 1979, by which time 1,986 had died.<sup>39</sup> Chrysotile was the only type of asbestos used in this plant except for two well-defined periods prior to 1949 when crocidolite was used. Regular measurements were introduced in the plant only in 1967 and fibre concentrations in earlier years were estimated by attempting to reproduce earlier working conditions. Given the passing of the 1931 Asbestos Industry Regulations and ventilation improvements in the plant, it was estimated that all the jobs within the plant except grinding and fibre preparation had exposures under 5 f/cc. By the 1950s, all exposures in the plant were estimated to be below 5 f/cc; and by 1969, pursuant to the then new regulations, exposures were reduced to less than 2 f/cc.

Table 5.2 indicates observed and expected mortality among men and women in the cohort after 10 years from first exposure to asbestos. Apart from 10 pleural mesotheliomas (and one further since the mortality analysis was completed), there was no evidence of any excess mortality among these workers either from all causes or from any particular cause including lung cancer (although it should be observed that the minimum period of follow-up was only 10 years).<sup>40</sup> And while a considerable number of the workers were employed for a short period of time, even those with the longest service, 20 or more years, and who were thus more heavily exposed, demonstrated no excess disease.<sup>41</sup> (Table 5.3 shows observed and expected mortality among those workers who had been employed at least 10 years.) As the authors commented, however, a noteworthy aspect of the study was the low exposures experienced by the workforce.<sup>42</sup> This occurred in part because of the number of short-term employees but was also a consequence of environmental control in the factory over an extended period of time. As a result, it is estimated that only 5% of the men employed after 1941 had a cumulative exposure of 100 fibres per cubic centimetre-years (f/cc-yrs). At 100 f/cc-yrs, Dr. J.C. McDonald estimated that the SMR for lung cancer

<sup>39</sup>A further 187 deaths occurred in this cohort in the 18-month period commencing January 1, 1980, yielding a total of 2,173 deaths. Of these additional 187 deaths, 13 were due to lung cancer, and one man was certified as dying of a pleural mesothelioma. See Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," p. 3.

<sup>40</sup>Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," pp. 1-3.

<sup>41</sup>Ibid., p. 3. In September 1983, as we were completing our Report, Dr. Murray M. Finkelstein and Mr. Robert A. Kusiak released in draft the results of a mortality study they had conducted of former employees of the Bendix Automotive Corporation located at Windsor, Ontario. These employees were potentially exposed to chrysotile asbestos. The authors noted that the results of their study are in line with those at Ferodo and concluded that workers exposed to asbestos in friction materials operations face health risks which are at worst marginal. See Murray M. Finkelstein and Robert A. Kusiak, "A Study of Mortality Among Employees of the Bendix Automotive Corporation, Windsor, Ontario," Toronto, Ontario Ministry of Labour, September 1983. (Mimeographed.)

<sup>42</sup>Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," p. 6.



**Table 5.2**  
**Observed and Expected Mortality After 10 Years from First Exposure**

Cause of Death	Number/Subject-Years			
	Men		Women	
	7,474/104,193		3,708/58,816	
	Obs	Exp	Obs	Exp
All causes	1,339	1,361.8	299	328.0
Lung and pleural cancer	151 (8)*	139.5	8 (2)	11.3
Gastrointestinal cancer	103	107.2	29	27.4
Other cancers	77	87.7	51	60.0
Other causes	1,008	1,027.4	211	229.3

**Note:** \*Number of pleural mesotheliomas included in parentheses.

**SOURCE:** Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 3 (Table 7).

Table 5.3  
Observed and Expected Mortality After Completing 10 Years' Employment

Follow-up After 10 Years' Exposure (Years) Number/Subject-Years	Men				Women			
	0-10		>10		0-10		>10	
	2,484/21,860		1,808/19,025		627/5,578		457/6,377	
Cause of Death	Obs	Exp	Obs	Exp	Obs	Exp	Obs	Exp
All causes	185	195.7	432	450.8	14	21.3	76	66.5
Lung and pleural cancer	23	21.3	58 (7)	47.4	0	0.7	2 (1)	2.2
Gastrointestinal cancer	23	16.3	25	35.8	0	1.8	8	5.7
Other cancers	7	12.6	21	28.2	3	4.5	14	10.7
Other causes	132	145.5	328	339.4	11	14.3	52	47.9

SOURCE: Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 4 (Table 8).

among his chrysotile miners was 104. In the Ferodo study of friction workers, the corresponding SMR was 106, and, as the authors pointed out, this slight excess could have arisen by chance; that is, it was not statistically significant.<sup>43</sup>

The 10 persons dying of mesothelioma in the main mortality study were examined in more detail in a case-control analysis. This showed that 8 of the 10 had definitely been exposed to crocidolite, and the authors concluded from their analysis (which sought to control for heavy chrysotile exposure) that crocidolite was predominantly responsible for the mesotheliomas.<sup>44</sup>

### (b) Connecticut

Very recently, Dr. A.D. McDonald and her colleagues have published the results of studies in three American asbestos factories in an effort to investigate the effect of fibre type and industrial process on asbestos-related disease.<sup>45</sup> Two of the factories manufactured textiles and will be discussed in this chapter under that heading. The third, located in Connecticut, manufactured friction products and packings using chrysotile only until 1957, when a small amount of anthophyllite was also used. A small amount of crocidolite was also used on an experimental basis, but not until 1964.<sup>46</sup>

The cohort examined was defined as all males who had been employed for one calendar month or more before January 1, 1959, and

<sup>43</sup>Ibid., pp. 6–7.

<sup>44</sup>Ibid., pp. 3–5. See also, Newhouse, Berry, and Skidmore, “A Mortality Study of Workers Manufacturing Friction Materials with Chrysotile Asbestos,” pp. 906–908. In his testimony before this Commission, Mr. Julian Peto suggested that there was a statistical error in this case-control analysis. Mr. Peto testified that the workers who were exposed to crocidolite in the area of the plant where crocidolite was used were almost exclusively production workers who had extremely heavy exposure to chrysotile. Mr. Peto suggested that what is missing from this study is an analysis that assumes that the chrysotile exposure caused the mesothelioma and an attempt to ascertain whether one could completely remove that association by allowing for crocidolite exposure. In Mr. Peto’s view, had that analysis been done, results symmetrical with those obtained by the authors, assuming that the crocidolite exposure caused the mesothelioma, would have been achieved. See RCA Transcript, Evidence of Mr. Julian Peto, 30 July 1981, Volume no. 25(B), pp. 83–86.

<sup>45</sup>Alison D. McDonald and John S. Fry, “Mesothelioma and Fiber Type in Three American Asbestos Factories — Preliminary Report,” *Scandinavia Journal of Work, Environment and Health* 8, Supp. 1 (1982): 53–58. See also, Alison D. McDonald et al., “Dust Exposure and Mortality in an American Chrysotile Textile Plant,” *British Journal of Industrial Medicine* 40 (1983): 361–367; Alison D. McDonald et al., “Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture,” *British Journal of Industrial Medicine* 40 (1983): 368–374; and Alison D. McDonald et al., “Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant,” *British Journal of Industrial Medicine*, in press, 1984.

<sup>46</sup>A. D. McDonald et al., “Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant.”

who had a social security number and name that matched with data in U.S. social security files. As defined, the cohort constituted 3,641 males, of whom over 96% were traced to the end of 1977, by which time 1,267 had died. Exposure information was limited, consisting in the main until the 1970s of insurance company surveys. Before 1970, measurements were of dust, not fibres, and the authors did not attempt to convert the particle measurements to fibres due largely to their lack of confidence in the results.<sup>47</sup>

The authors did estimate that in the 1930s dust levels were generally in the range of 1 to 5 mppcf, save for certain much dustier processes (fibre preparation, fibre mixing, grinding, and finishing) where the levels were considerably higher. The average length of employment in this plant was 8.7 years, and the average dust concentration to which the men were exposed was 1.84 mppcf.<sup>48</sup> The exposure levels appear to have been quite similar to those at Ferodo.<sup>49</sup> Table 5.4 shows male deaths 20 years after first employment, by cause, in relation to duration of service.

The results of the study indicated an overall SMR of 108.5 (using Connecticut mortality rates), but the excess was not found in the longer-service employees. It occurred predominantly among those who had worked for one year or less. SMRs were increased for lung and gastrointestinal cancer, but again this was largely due to the excess in men employed less than one year. This lack of any systematic dose-response relationship in the study was also present when the data were analyzed by cumulative dust exposure, although there was some suggestion of an increasing risk for lung cancer with increasing exposure if the lowest dust category were excluded. No case of mesothelioma was found within this cohort.<sup>50</sup>

The authors of the study suggested that the excess mortality from all causes of death including lung cancer in the men employed less than one year may have arisen from the poorer health of those short-term employees and that otherwise the results suggest that the adverse health effects of employment in this chrysotile friction products plant were quite small.<sup>51</sup> While not as negative as the results at Ferodo, they are certainly compatible with those results and they are quite similar to the experience of the Quebec chrysotile miners.

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<sup>47</sup>Ibid., pp. 4–5.

<sup>48</sup>Ibid., pp. 5–6.

<sup>49</sup>Ibid., p. 9. The authors have stated that “The exposure levels in the two factories appear to have been fairly similar.”

<sup>50</sup>Ibid., p. 7.

<sup>51</sup>Ibid., p. 8.



Table 5.4  
Male Deaths 20 Years After First Employment, by Cause, in Relation to Duration of Service

Cause of Death*	Length of Gross Service (Years)								Complete Cohort	
	<1		1<5		5<20		≥20			
	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR
All causes	246	129.9	189	104.0	130	104.8	238	97.2	803	108.5
Malignant neoplasms	60	144.9	50	125.7	29	114.0	63	118.3	202	126.5
Respiratory	24	180.0	19	149.4	9	122.6	21	133.4	73	148.7
Digestive	17	132.9	16	128.3	5	60.6	21	116.9	59	114.4
Other	19	120.4	25	164.9	15	190.4	21	107.2	70	115.9
Heart disease	99	125.3	79	104.9	44	83.7	100	93.1	322	102.5
Respiratory tuberculosis	0	—	0	—	0	—	4	283.3	4	145.9
Other respiratory	13	196.3	8	126.2	4	85.2	8	92.2	33	—
Pneumoconiosis	(6)	—	(3)	—	(1)	—	(2)	—	(12)	—
Cerebrovascular	18	137.6	14	108.4	15	142.7	20	102.4	67	119.6
Accidents	11	121.1	5	69.2	5	101.7	7	68.6	28	89.1
Other known	37	123.5	24	90.4	29	147.1	35	87.9	125	107.7

**Note:** \*ICD [International Classification of Diseases] Codes, except that ICD Codes 160-164 are here grouped under "respiratory" malignant neoplasms and the "other respiratory" category includes only bronchitis, pneumonia, and pneumoconiosis (ICD 490-502, 523-524).

SOURCE: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant," *British Journal of Industrial Medicine*, in press, 1984, Table 4.

### B.3 Asbestos Manufacturing — Textiles

#### (a) Rochdale, England

No group of asbestos workers has been more studied than those employed at the Rochdale textile factory in England. Rochdale was the subject of the first systematic epidemiological study of asbestos-related disease published by Sir Richard Doll in 1955.<sup>52</sup> The health experience of the workers at Rochdale formed the basis of the 1968 report of the British Occupational Hygiene Society which led to the adoption of the 2 f/cc standard in the United Kingdom.<sup>53</sup> The morbidity experience of these workers has continued to be studied by Mr. Geoffrey Berry and his colleagues,<sup>54</sup> while their mortality experience has been recently reviewed by Mr. Julian Peto.<sup>55</sup> We have commented elsewhere in this Report on Mr. Berry's asbestosis investigation at Rochdale.<sup>56</sup> Here we focus on Mr. Peto's mortality investigation.

We observe that both the type of asbestos used at Rochdale and the exposure information have been the subject of debate. There is no doubt that chrysotile asbestos has been the predominant fibre type at the plant, but there is uncertainty over the extent to which crocidolite was also used. It would, however, appear that at least some crocidolite was utilized at

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<sup>52</sup>Richard Doll, "Mortality from Lung Cancer in Asbestos Workers," *British Journal of Industrial Medicine* 12 (1955): 81–86. A total of 113 workers exposed to asbestos for at least 20 years were followed up; 39 deaths occurred in the group versus 15.4 expected. The excess was due to excess deaths from lung cancer (11 versus 0.8 expected) and from other respiratory and cardiovascular diseases (20 versus 7.6 expected). *Ibid.*, Table 4, p. 84.

<sup>53</sup>British Occupational Hygiene Society, Committee on Hygiene Standards, "Hygiene Standards for Chrysotile Asbestos Dust," *Annals of Occupational Hygiene* 11 (1968): 54–58 [Appendix (i)]. See also, Chapter 3, Section B of this Report.

<sup>54</sup>Geoffrey Berry and Hilton C. Lewinsohn, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part I. Morbidity," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 185–194; and Geoffrey Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," *British Journal of Industrial Medicine* 36 (May 1979): 98–112.

<sup>55</sup>Julian Peto et al., "A Mortality Study Among Workers in an English Asbestos Factory," *British Journal of Industrial Medicine* 34 (1977): 169–173; Julian Peto, "The Hygiene Standard for Chrysotile Asbestos," *The Lancet* 1 (4 March 1978): 484–489; Julian Peto, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part II. Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 195–201; Julian Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 703–711; and Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 829–836.

<sup>56</sup>See Chapter 2, Section D.1 of this Report.

Rochdale even as late as 1969, although the amount is not accurately known.<sup>57</sup>

In studies of Rochdale published prior to 1979, exposures were calculated on the basis of area samples. Area thermal precipitator measurements in millions of particles per cubic foot were taken between 1951 and 1960 and were converted to fibres per cubic centimetre using the observed ratio of parallel measurements obtained by the two methods in 1960 and 1961. From 1961 onwards, fibre levels were estimated in each area from actual membrane filter measurements. There were no routine measurements in the period 1933–1951, and exposures during this period were estimated to be as much as 50% higher than those in the early 1950s. On the basis of these data, it was estimated that the mean dust level at Rochdale was 10.8 f/cc in 1951, falling to 2.9 f/cc by 1972.<sup>58</sup>

Then in the late 1970s, the hygiene officers at Rochdale undertook a complete review of the exposure estimates at the plant.<sup>59</sup> Taking into consideration the improvements in fibre counting techniques since 1961,<sup>60</sup> the new survey produced substantially higher estimates of exposure levels than

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<sup>57</sup>Mr. Peto has indicated that the asbestos exposure at Rochdale was predominantly to chrysotile, although a small proportion of crocidolite fibre was used at various times after 1933. See Peto, "The Hygiene Standard for Chrysotile Asbestos," p. 487. In a more recent publication, Mr. Peto observed that the extent of exposure to crocidolite at Rochdale is not accurately known, but appears to have been much lower than that at the East London asbestos factory studied by Newhouse and Berry. See Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," p. 708. In their report to the U.K. Advisory Committee on Asbestos, Dr. E. Donald Acheson and Dr. Martin J. Gardner stated that "There appears to be uncertainty and possibly a conflict of evidence about the extent and period of usage of crocidolite at Rochdale which the present authors have been unable to resolve. . . . A recent communication from Mr. H.D.S. Hardie of Turner and Newall states that between 1931 and 1970 not less than 2,500 tonnes [metric tons] of crocidolite were used at Rochdale, i.e. an average of about 60 tonnes per annum." See Acheson and Gardner, "The Ill Effects of Asbestos on Health," paragraph 119, p. 26. However, in their 1983 update, the authors now suggest that about 10,000 tonnes of crocidolite fibre in yarn were used at Rochdale between 1931 and 1970. See Acheson and Gardner, *Asbestos: The Control Limit for Asbestos*, paragraph 30, p. 5.

<sup>58</sup>Peto et al., "A Mortality Study Among Workers in an English Asbestos Factory," pp. 171–172; and Peto, "The Hygiene Standard for Chrysotile Asbestos," p. 487.

<sup>59</sup>Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," pp. 831–832.

<sup>60</sup>Mr. Peto has observed that the most notable improvement in counting technique was the change to graticule counting, thereby increasing the estimated fibre count by a factor of 2 or more. See Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," p. 831. Dr. Acheson testified that in his view the U.K. Advisory Committee on Asbestos was prepared to accept the fact that there had been approximately a fivefold increase in the rigorousness of the standard in the United Kingdom due to modern counting techniques (graticule vs. full view) and due to the difference between personal and area sampling. RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 55.

previously reported and indicated that average dust levels were in the region of 30 f/cc in 1951 and remained high until 1970.<sup>61</sup>

While the estimates of exposure have changed over the years at Rochdale and while average rather than individual estimates have been made, still this is the only asbestos manufacturing plant studied which has fibre measurements sufficiently far back in time to provide direct quantitative data that can be used for epidemiological study and risk assessment.<sup>62</sup>

The most recent mortality studies of Rochdale were published by Mr. Julian Peto in 1980. His total cohort of 1,106 workers was broken down into five sub-cohorts based on whether a worker was first exposed prior to 1933 or after 1933 and whether he worked more than 10 or more than 20 years at the plant. The workers first exposed after 1933 were then further subdivided between those first starting employment between 1933 and 1950 and those first starting in 1951 or later. The 679 men who entered the factory in 1933 or later and had by the end of 1972 been exposed for at least 10 years were followed to the end of 1978.<sup>63</sup> Table 5.5 shows the mortality experience of these 679 male asbestos textile workers.

The SMR for lung cancer, 20 years or more from first exposure, for all persons who worked at Rochdale for at least 10 years starting in 1951 or later, was approximately 500 (8 lung cancer deaths versus 1.62 expected).

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<sup>61</sup>Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," p. 832. The revised estimates of mean dust levels at Rochdale indicate figures of 32.4 f/cc in 1951; 23.9 f/cc in 1956; 12.2 f/cc in 1961; 12.7 f/cc in 1966; and 4.7 f/cc in 1971. The corresponding previous estimates were 10.8 f/cc in 1951; 5.3 f/cc in 1956; 5.2 f/cc in 1961; 5.4 f/cc in 1966; and 3.4 f/cc in 1971.

<sup>62</sup>Mr. Peto indicated in his evidence that there have been two revisions of the dust estimates at Rochdale since the publication of the 1968 British Occupational Hygiene Society report. There was the revision which led to the analysis by Berry et al. in "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory"; and the further revision, which was given to Mr. Peto and upon which he based his 1980 publication called "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory." See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 90-91. See also, British Occupational Hygiene Society, Committee on Hygiene Standards, "Hygiene Standards for Chrysotile Asbestos Dust," Appendix (i), pp. 54-58. Mr. Peto maintained in his testimony that in his view there was no other cohort study which had as reasonably reliable evidence in terms of fibres and as moderately good measurements as the Rochdale cohort. RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 83. Dr. Finkelstein testified that in his view Mr. Peto's study, which dealt solely with lung cancer and had one data point, was the only one which provided any data for a quantitative risk assessment. See RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 112-113.

<sup>63</sup>Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," pp. 829-830. The breakdown of the entire cohort of 1,106 workers was described in an earlier article by Peto et al., "A Mortality Study Among Workers in an English Asbestos Factory," p. 170.



Table 5.5  
Mortality Experience of 679 Male Asbestos  
Textile Workers

Year First Exposed	Period Since First Exposure (Years)	Man-Years	Lung Cancer		Pleural Mesothelioma		Other Cancers		Asbestosis		Other Respiratory Disease		Other Causes	
			O	E	O	Rate x 10 <sup>3</sup>	O	E	O	Rate x 10 <sup>3</sup>	O	E	O	E
1933- 1950  n = 424	10-	1,633	2	1.80	0	0.0	1	2.73	0	0.0	1	2.38	8	11.02
	15-	1,860	4	2.98	0	0.0	2	4.06	2	1.1	3	3.73	17	16.71
	20-	1,760	3	3.97	1	0.6	6	5.16	2	1.1	4	5.02	29	21.64
	25-	1,496	10	4.54	2	1.3	6	5.98	2	1.3	9	6.08	24	25.20
	30-	837	8	3.14	2	2.4	5	4.21	2	2.4	4	4.47	16	17.80
	35+	507	1	2.20	2	3.9	4	2.93	2	3.9	5	3.06	12	12.33
Total			28	18.63	7	—	24	25.07	10	—	26	24.73	106	104.70
1951 or later  n = 255	10-	1,123	1	1.30	0	0.0	0	1.62	0	0.0	1	1.17	5	6.49
	15-	1,022	3	1.74	0	0.0	3	2.16	0	0.0	2	1.63	8	8.66
	20-	556	7	1.31	0	0.0	2	1.64	0	0.0	0	1.30	4	6.61
	25+	96	1	0.31	0	0.0	0	0.37	0	0.0	0	0.32	1	1.52
	Total	2,797	12	4.65	0	—	5	5.80	0	—	3	4.42	18	23.28

SOURCE:

Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J. C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), Table 1, p. 830.

The average cumulative exposure for this group of employees was 100 f/cc-yrs in terms of the earlier exposure estimates and in the order of 300 f/cc-yrs in terms of the revised estimates.<sup>64</sup> Using these revised estimates, the SMR for lung cancer at 100 f/cc-yrs was approximately 165 (compared to 104 for the Quebec miners and 106 for the Ferodo friction workers). While admittedly the numbers are small, the mortality risk for Rochdale textile workers appears to be considerably higher than for chrysotile miners or friction product workers.

Mr. Peto has reported 14 cases of pleural mesothelioma at Rochdale among those first employed prior to 1951;<sup>65</sup> 7 of these cases occurred among those first employed prior to 1933. Among those first employed after 1933 but before 1951, the observed incidence of mesothelioma rose steadily from 60 per 100,000 per annum at 20 to 25 years after first employment to 400 per 100,000 per annum beyond 35 years.<sup>66</sup> Due to the uncertainty over the use of crocidolite at Rochdale, it was not possible to determine the type of fibres to which the mesothelioma cases were exposed. Mr. Peto has, however, commented that in his view the occasional exposure to crocidolite was unlikely to be the major cause of the incidence of mesothelioma at Rochdale.<sup>67</sup>

### **(b) Charleston, South Carolina**

In September 1980, Dr. John M. Dement, an industrial hygienist at the time with NIOSH in the United States, presented findings of a study of asbestos textile workers at Charleston, South Carolina, which greatly surprised everyone familiar with the literature on asbestos.<sup>68</sup> The surprise was generated by two considerations: the excess disease incidence at Charleston was orders of magnitude higher than had been found in any other reported study, and yet the exposure of the workers had been to chrysotile asbestos.

The cohort reported on by Dr. Dement was limited to the 768 white males employed 6 or more months in the textile production operations of the Charleston plant with at least one month of employment between

<sup>64</sup>RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 103.

<sup>65</sup>Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," p. 703.

<sup>66</sup>Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," p. 831.

<sup>67</sup>Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," p. 708. See, however, note 57, *supra*.

<sup>68</sup>John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1-4 (1982): 869-887. This paper was first presented at the Fifth International Symposium on Inhaled Particles at Cardiff, Wales, in September 1980. See also, John M. Dement et al., "Exposures and Mortality Among Chrysotile Asbestos Workers. Part I: Exposure Estimates," *American Journal of Industrial Medicine* 4 (1983): 399-419.

January 1, 1940 and December 31, 1965. The cohort was followed through 1975, thus ensuring a minimum latency period of 10 years.<sup>69</sup>

There were 5,952 measurements collected in the period 1930–1975, although all but 376 were collected after 1959. Prior to 1965, all measurements were in dust particles by impinger; from 1965 to 1971, fibre measurements by membrane filter were also made; and, in 1971, the impinger method was entirely replaced by the membrane filter method. Dr. Dement converted his dust particle counts to fibre counts on the basis of two sources of data: 120 paired impinger membrane filter samples collected in the plant by the U.S. Public Health Service in 1965 and 986 paired samples collected in the plant in the period 1968–1971. On the basis of these data Dr. Dement used a conversion factor of 3 f/cc to 1 mppcf for all textile operations save preparation, for which a conversion factor of 8 was used.<sup>70</sup>

It is clear that chrysotile was the only asbestos received at Charleston as a raw fibre. Crocidolite was used in weaving from the 1950s until approximately 1975, but the amount was trivial, the weaving was done by a wet process, and crocidolite was never carded, spun, or twisted.<sup>71</sup>

Table 5.6 sets out the observed and expected deaths, by cause, for the white male asbestos textile workers in the cohort. In the entire cohort there were 191 observed deaths versus 141.84 expected, for an SMR of 135; there were 26 lung cancer deaths versus 7.47 expected, for an SMR of 348; and there were 15 deaths from asbestosis. Smoking data tended to show that the prevalence of smoking among men in the cohort was nearly identical to that of U.S. white males as a whole.<sup>72</sup>

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<sup>69</sup>Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," p. 870.

<sup>70</sup>Ibid., pp. 871–872; and Dement et al., "Exposures and Mortality Among Chrysotile Asbestos Workers. Part I: Exposure Estimates," pp. 408–409.

<sup>71</sup>Dement et al., "Exposures and Mortality Among Chrysotile Asbestos Workers. Part I: Exposure Estimates," p. 400. The authors have indicated that the total amount of crocidolite ever processed at the Charleston plant was less than 2,000 pounds. They also stated that according to company personnel, an average of approximately 6 to 8 million pounds of chrysotile was processed annually. See also, RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, pp. 91–100. Dr. Dement also observed in his evidence that exposure to crocidolite could not have had a significant effect on lung cancer mortality in that almost all of the lung cancer deaths occurred 30 or more years from first exposure.

<sup>72</sup>Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," pp. 880–881. Among the asbestos workers for whom smoking data were available, 52.4% were found to be current smokers, 25.3% were non-smokers, and 22.3% were past smokers. The corresponding figures for U.S. white males in 1965 were 51.5%, 26.4%, and 22.1%.

**Table 5.6**  
**Observed and Expected Deaths, by Cause, for White Male**  
**Asbestos Textile Workers, 1940-1975**

Cause of Death	ICDA 7th	Obs	Exp	SMR
	List Number			
All causes		191	141.84	135†
Malignant neoplasms		43	24.66	174†
Digestive system	150-159	9	7.10	127
Trachea, bronchus, and lung	162-163	26	7.47	348†
Other and unspecified sites		8	10.09	79
Disease of the central nervous system	330-334, 345	9	7.72	117
Disease of the circulatory system	400-468	58	57.43	101
All tuberculosis	001-019	4	2.77	144
Non-malignant respiratory disease		18	6.85	263†
Acute upper respiratory infection	470-475	0	0.04	—
Influenza	480-483	0	0.37	—
Pneumonia	490-493	0	2.98	— *
Bronchitis	500-502	0	0.41	—
Other respiratory diseases	510-527	18	3.05	590†
Accidents	800-962	14	16.64	84
Other violent deaths	963-964	7	6.10	115
	970-985			
All other known causes		23	17.92	128
Unknown causes		15	1.75	—

**Notes:** \*Significant at  $p < 0.05$ .

†Significant at  $p < 0.01$ .

SOURCE: John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1-4 (1982): 873 (Table 3).



Table 5.7 is a summary of dose-response relationships for selected causes of death. Dose-response analysis was limited to those workers who had achieved 15 or more years since first employment. Cumulative exposure was divided into four exposure categories, and for lung cancer the SMR rose from 223 in the lowest category to 1,553 in the highest, with an overall SMR of 399. Corresponding overall SMRs for deaths from all causes and from non-malignant respiratory disease were 151 and 642 respectively. Despite the extraordinarily elevated lung cancer risk, some 50 times greater than that found among friction product workers or chrysotile miners, there was only one mesothelioma death (peritoneal) in this cohort, and the incidence of gastrointestinal cancer was only slightly increased, with an SMR of 127.

Dr. Dement's study has been subjected to rigorous scrutiny by many of the world's acknowledged experts on asbestos in an effort to explain and qualify his findings.<sup>73</sup> Several concerns about his study were voiced during our hearings by other witnesses. We list these concerns and comment on them as follows:

(i) The Charleston workers may have had prior shipyard exposure.<sup>74</sup> While Dr. Dement did not have a prior occupational history on his entire cohort, he did have past employment histories on 10 of the 26 lung cancer cases and none of them had shipyard experience. Furthermore, the Charleston shipyard used crocidolite and Dr. Dement observed that if a high proportion of his textile workers had been employed in the shipyards, he would have expected to see more than one mesothelioma.<sup>75</sup>

(ii) There were few measurements prior to 1960.<sup>76</sup> While this is obviously true, compared to other studies the quantity of these measurements is appreciable. Moreover, textile operations have not changed a great deal over time, and according to Dr. Dement's evidence before this Commission, insurance company measurements, government measurements, and the company's own measurements were quite consistent.<sup>77</sup>

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<sup>73</sup>For example, Dr. Gibbs testified that he and a number of other scientists met with Dr. Dement to look at the Charleston data to determine whether there was any explanation as to why the risks in relation to exposure appeared to be so different from those found in all other studies. See RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 60-61.

<sup>74</sup>See, for example, RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 45.

<sup>75</sup>RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 12.

<sup>76</sup>For example, Mr. Peto testified that the Dement study suffered from the fact that most of the excess mortality was in workers who were first exposed in the 1930s and 1940s when no reliable measurements were taken. See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 145.

<sup>77</sup>RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, pp. 14-15.

Table 5.7  
Summary of Dose-Response Relationships, for Selected Causes, Among White Males Achieving 15 or More Years' Latency

Cumulative Dose (Fibre cm <sup>3</sup> -3 Days)	All Causes			Diseases of the Circulatory System (400-468)			Lung Cancer (162, 163)			Digestive System Cancer (150-159)			Other Non-malignant Respiratory Diseases (510-527)		
	Obs	Exp	SMR	Obs	Exp	SMR	Obs	Exp	SMR	Obs	Exp	SMR	Obs	Exp	SMR
<10,000	65	55.17	118*	21	24.37	86	8	3.59	223*	1	2.73	37	1	1.41	71
10,000-40,000	57	32.83	174†	23	16.94	136*	7	1.96	357†	3	1.85	162	7	0.78	897†
40,000-100,000	32	13.63	235†	7	6.37	110	9	0.92	978†	3	0.79	380	7	0.38	1,842†
100,000-200,000	6	3.42	175	1	1.30	77	2	0.13	1,553*	0	0.16		2	0.08	2,500*
Total	160	105.05	151†	52	48.98	106	26	6.51	399†	7	5.53	127	17	2.65	642†

Notes: \* Significant at  $p < 0.05$ .

† Significant at  $p < 0.01$ .

SOURCE: John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1-4 (1982): 877 (Table 7).

(iii) The estimated exposures were far too low in light of the pictorial evidence of prior conditions in the plant.<sup>78</sup> This factor is difficult to assess, but we note that Dr. A.D. McDonald, who has studied the same plant using the same exposure information, indicated that the estimates were reasonably consistent among several groups of observers and similar to levels in other American textile plants and probably also to Rochdale.<sup>79</sup>

(iv) Dr. Dement's conversion ratios from particles to fibres were too low, thereby overestimating the risk.<sup>80</sup> It does appear that the conversion factors used by Dr. Dement were on the low side of the range of conversions done by Ayer, Lynch, and Fanney for the U.S. Public Health Service in 1965.

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<sup>78</sup>In his testimony, Dr. Hans Weill indicated that Dr. Dement himself had shown a slide of a carding machine in the Charleston plant in the 1930s, and the slide indicated that conditions were very very dusty. Yet, according to Dr. Weill, in the published paper Dr. Dement assessed the average exposure at that time in the particular job as 5 f/cc, which Dr. Weill found a little hard to believe. See RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 105–106. Dr. A.D. McDonald, who has studied the same plant, observed in her testimony that the quantitative data on past exposure is quite perplexing in that they are relatively low compared to what one might have expected from some of the pictorial evidence. See RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 25, 62. In their paper, Dr. A.D. McDonald and her colleagues discussed the possibility that both Dr. Dement and her team had failed to give sufficient weight to the occasional extremely high exposure which, from all accounts, many employees experienced during overtime work. In the view of Dr. A.D. McDonald and her colleagues, these casual exposures, although significant, were unlikely to explain a more than twofold error in cumulative exposure estimates. See A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," p. 366.

<sup>79</sup>A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," p. 366.

<sup>80</sup>For example, Dr. Weill suggested in his evidence that a conversion factor of 3 was a little too low because the simultaneous samplings of the U.S. Public Health Service suggested at least a 6 to 1 ratio if not higher for textile manufacturing. Dr. Weill observed that in textile manufacturing asbestos is a very much higher proportion of the total dust than, for example, in asbestos-cement manufacturing. See RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 101–102. Dr. Gibbs also felt that Dr. Dement's conversion ratios might have been a little lower than he would have expected. See RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 60–61. In their own review of the environmental measurements, Dr. A.D. McDonald and her colleagues observed a conversion range of 1.3 to 10.0, with an average of about 6, which was the same figure that Ayer, Lynch, and Fanney of the U.S. Public Health Service concluded might be appropriate for textile processes. See A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," p. 366. See also, Howard E. Ayer, Jeremiah R. Lynch, and Julius H. Fanney, "A Comparison of Impinger and Membrane Filter Techniques for Evaluating Air Samples in Asbestos Plants," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 274–287.

Nonetheless, both Mr. Howard E. Ayer himself and Dr. A.D. McDonald considered the conversion factors used by Dr. Dement to be reasonable.<sup>81</sup>

(v) Dr. Dement has used an unorthodox methodology to calculate his exposure-response relationships.<sup>82</sup> As Dr. Murray M. Finkelstein observed, it would appear that the methodology used by Dr. Dement tended to underestimate the risk at low exposure categories and overestimate the risk at higher exposure categories. Still, his methods do not detract from the overall results,<sup>83</sup> and, as Dr. Finkelstein also observed, Dr. Dement's study appears to be a thoughtful and intelligent approach to the problem of exposure assessments.<sup>84</sup>

(vi) Dr. Dement used national mortality rates for his control population whereas had he used local mortality rates, his excess mortality would have been less since Charleston and the surrounding counties have a lung cancer

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<sup>81</sup> See RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, p. 21; and RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 60–61. Dr. J.C. McDonald testified that Dr. Dement is an occupational hygiene engineer and is probably one of the most competent people attempting to do conversions from particles to fibres. See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 59. Dr. Dement, in his own testimony, admitted that his conversions were on the low side of the range of conversions done by Ayer, Lynch [and Fannery] for the U.S. Public Health Service. However, Dr. Dement's conversions were certainly within the range of those conversions. See RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 33.

<sup>82</sup> Dr. Finkelstein testified that Dr. Dement and his colleagues had adopted a method whereby workers move from one exposure category to the next as exposure accumulates. However, person-years of risk are left behind in former categories and begin to be freshly contributed to the new category as it is entered. Dr. Finkelstein suggested that this is not an appropriate method when one is dealing with mortality from diseases of long latency. According to Dr. Finkelstein, as a result of using this methodology, too many deaths and too few person-years of risk would be attributed to the higher exposure categories and too few deaths to the lower exposure categories. The net result is an overestimate of the slope of the dose-response curve. See RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 113–116. See also, letter from Dr. Murray M. Finkelstein to the Editor, *American Journal of Industrial Medicine*, 27 May 1983. This letter, which contains Dr. Finkelstein's critique of the methodology used by Dr. Dement and his colleagues, is to be published in the *American Journal of Industrial Medicine*.

<sup>83</sup> Dr. Dement, in his testimony, indicated that his methodology did tend to underestimate the risk at lower levels and overestimate it at higher levels, but only slightly. The alternative method, for example, used by Dr. Finkelstein in estimating dose, would be to have a person accumulate exposure up to a fixed point, say, 20 years, cut off cumulative exposure at that point, and then have a follow-up period. According to Dr. Dement, each of these two methods has its own advantages. See RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, pp. 29–31.

<sup>84</sup> RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 55.



mortality rate considerably above the national average.<sup>85</sup> Dr. Dement discussed his choice of national rates in his published report. He rejected the use of local rates because many citizens working in the local shipyard industry would have been exposed to asbestos and because the Charleston plant itself would have contributed to the high local cancer rate. However, even if lung cancer mortality rates for the contiguous counties were used, expected mortality rates would increase by only 15%, not enough to account for the observed excess in the cohort.<sup>86</sup>

(vii) Dr. Dement has not included black males in his cohort. He has not done so apparently because of the poor trace rate and cause of death information for this group. Still, as Dr. Dement has pointed out, at least in terms of duration of employment, the black male employees have elevated SMRs fairly consistent with the white male data.<sup>87</sup>

We have devoted considerable space to a discussion of the Charleston study. We have done so because of the serious implications raised by its startling results. In the end, we are driven to the general conclusion that some or all of the various comments or criticisms which have been made of the study may account for part of the difference between it and other studies, but even together they cannot account for all of the difference.<sup>88</sup>

Cogent confirmation of Dr. Dement's findings may be found in a subsequent and independent investigation of the health experience at the Charleston plant by Dr. A.D. McDonald and her associates.<sup>89</sup> Their cohort was considerably larger than Dr. Dement's, comprising in all 2,543 men employed for at least one month between 1938 and 1958 and followed to the end of 1977, by which time 863 employees or 34% of the cohort had died. Of the 863 deaths, there were 6 cases for which the age at death was not known. The authors' man-years calculations were, therefore, based

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<sup>85</sup>RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 36-37. Lung cancer death rates for South Carolina were nearly equal to U.S. rates. On the other hand, rates for the county in which the Charleston plant was located were 75% higher than U.S. rates for white males.

<sup>86</sup>Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," pp. 879-880.

<sup>87</sup>See RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 81. Dr. Dement also observed in his evidence that the entire population of workers at the Charleston plant was approximately 10,000, but that included both textile and non-textile workers, black and white, male and female workers. See *ibid.*, p. 40.

<sup>88</sup>Dr. A.D. McDonald reached similar conclusions. RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, p. 63. In their paper, Dr. A.D. McDonald and her colleagues suggested that even giving effect to all of the critiques, it was improbable that the fiftyfold difference in exposure-response between the Quebec miners cohort and the Charleston cohort could be reduced to less than about tenfold. See A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," p. 366.

<sup>89</sup>A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant."

upon 857 deaths, and this figure is used in Table 5.18 in this chapter. In calculating SMRs, only deaths 20 or more years from first employment were included. Table 5.8 shows male deaths 20 years after first employment, by cause, in relation to duration of service. For the entire cohort as defined, the SMR for all causes of death was 127.4; for lung cancer, 199.5; and for abdominal cancer, 151.7 (the latter figure being higher than Dr. Dement's corresponding figure for gastrointestinal cancer). There were 20 deaths from pneumoconiosis, all among men with 20 or more years' employment, and there was only one mesothelioma death in the entire cohort, the same case reported by Dr. Dement.<sup>90</sup> When calculated according to cumulative dust exposure, the SMR for lung cancer rose sharply, reaching 1,031.9 (on the basis of 8 cases) in the highest dust category, and the relationship was essentially linear. Table 5.9 sets out male deaths 20 years after first employment, by cause, in relation to dust exposure accumulated up to 10 years before death.

Considering that Dr. Dement excluded from his cohort clerical employees and employees who had worked less than 6 months, whereas Dr. A.D. McDonald did not, the findings of the two studies are almost identical.<sup>91</sup> In seeking to compare them with the quite different results in chrysotile mining and friction products manufacturing, two further observations may be made. First, the average dust concentrations at Charleston, although low when compared with mining and milling, were in the same range as the Connecticut friction products plant in which little or no excess lung cancer mortality occurred — 1.80 mppcf compared with 1.84 mppcf.<sup>92</sup> Second, there were no major differences in smoking habits as between the textile workers and the Quebec miners and millers that might explain the different incidence of lung cancer in the two cohorts.<sup>93</sup>

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<sup>90</sup>Ibid., p. 365. The one death was a man born in 1904 who died in 1967. He was first employed at the plant in 1925, worked as a mule spinner from 1933 to 1955 and as an oven helper until he left in 1965. There was no autopsy. In her testimony, Dr. A.D. McDonald indicated that she had considered whether there were cases of mesothelioma occurring at Charleston that had not been detected. She observed that the Department of Pathology at the Medical College of South Carolina at Charleston is very interested in mesothelioma and has been looking very hard for cases and has yet not found any save for the one case reported by her and by Dr. Dement and one other case that did not appear in either cohort. See RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 48–49.

<sup>91</sup>A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," p. 366 and Figure 2, p. 366.

<sup>92</sup>Ibid., Table 3, p. 363; and A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant," Table 3.

<sup>93</sup>A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," pp. 366–367. Dr. A.D. McDonald and her colleagues noted that the dose-response line at Charleston is 50 times steeper than the dose-response line for the Quebec mining cohort.

Table 5.8  
Male Deaths 20 Years After First Employment, by Cause, in Relation to Duration of Service

Cause of Death*	Length of Gross Service (Years)								Complete Cohort	
	<1		1<5		5<20		≥20			
	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR
All causes	159	107.4	113	122.7	120	156.1	178	136.7	570	127.4
Malignant neoplasms										
Respiratory	8	78.2	10	163.9	15	304.1	26	317.3	59	199.5
Abdominal	6	107.9	5	146.4	7	240.3	8	151.4	26	151.7
Other	12	130.2	7	124.9	9	195.9	7	46.2	35	127.5
Heart disease	69	108.9	34	87.6	45	141.7	70	120.8	218	113.7
Respiratory tuberculosis	1	231.8	1	347.8	1	307.9	1	131.5	4	222.8
Other respiratory	3	53.3	3	85.6	2	78.3	27	557.5	35	207.3
Pneumoconiosis	(0)	—	(0)	—	(0)	—	(20)	—	(20)	—
Cerebrovascular	9	83.0	14	193.0	6	107.3	9	76.2	38	107.2
Accidents	18	121.2	8	89.7	5	75.8	9	85.0	40	97.0
Other known	30	116.9	28	175.5	23	177.7	21	92.3	102	132.4
Not known	3		3		7		0		13	

**Note:** \*ICD Codes, except that ICD Codes 160-164 are here grouped under "respiratory", malignant neoplasms and the "other respiratory" category includes only bronchitis, pneumonia, and pneumoconiosis (ICD 490-502, 523-524).

SOURCE: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," *British Journal of Industrial Medicine* 40 (1983): 364 (Table 4).

Table 5.9

Male Deaths 20 Years After First Employment, by Cause, in Relation to Dust Exposure  
(Million Particles Per Cubic Foot-Years) Accumulated to 10 Years Before Death

Cause of Death*	Dust Exposure (mppcf-yrs)									
	<10		10<20		20<40		40<80		≥80	
	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR
All causes	376	115.5	55	125.5	63	156.9	43	170.8	33	264.4
Malignant neoplasms										
Respiratory	31	143.1	5	182.7	8	304.2	7	419.5	8	1,031.9
Abdominal	14	114.9	4	231.6	4	247.0	4	383.6	0	—
Other	28	140.0	3	109.2	1	44.9	0	—	3	383.5
Heart disease	143	103.5	28	143.6	29	166.6	10	88.6	8	149.9
Respiratory tuberculosis	3	264.4	0	—	0	—	1	634.4	0	—
Other respiratory	8	65.9	2	119.5	6	421.7	13	1,407.8	6	1,296.0
Pneumoconiosis	(0)	—	(0)	—	(3)	—	(9)	—	(8)	—
Cerebrovascular	29	115.3	2	50.0	4	124.4	2	93.4	1	99.8
Accidents	31	99.2	2	54.1	5	152.9	1	49.4	1	120.0
Other known	79	140.4	9	116.9	4	630	5	111.5	5	236.3
Not known	10		0		2		0		1	

**Note:** \*ICD Codes, except that ICD Codes 160-164 are here grouped under "respiratory" malignant neoplasms and the "other respiratory" category includes only bronchitis, pneumonia, and pneumoconiosis (ICD 490-502, 523-524).

SOURCE: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," *British Journal of Industrial Medicine* 40 (1983): 364 (Table 5).



**(c) Lancaster, Pennsylvania**

Dr. A.D. McDonald and her colleagues have also recently reported on another cohort of asbestos workers employed in a factory in rural Pennsylvania which manufactures mainly textile products but also some friction materials.<sup>94</sup> According to the authors, chrysotile was the principal type of asbestos used in the factory but crocidolite and amosite were also used from 1924 onwards, with the latter being utilized in particularly large quantities during World War II to make insulation blankets for the U.S. Navy.

This cohort was constituted in exactly the same way as the cohorts in the Charleston and Connecticut factory studies and in the Pennsylvania plant comprised 4,137 men, of whom 1,400 had died by the end of 1974. Dust levels at the plant appear to have been somewhat higher than in the Charleston or Connecticut plants, with average concentrations of 2.32 mppcf.<sup>95</sup> Table 5.10 shows male deaths 20 years after first employment, by cause, in relation to duration of service. The SMR for all causes of death, 20 years after first employment, was 109. The SMR for lung cancer among long-term employees was 158.9 and among those in the highest dust category was 416.1.<sup>96</sup> A comparison of the relative risks of death from all causes, from lung cancer, and from pneumoconiosis in the Charleston plant and in this Pennsylvania plant produced reasonably similar results.<sup>97</sup> What was not similar, however, was the mesothelioma incidence: in this plant, there were 14 deaths from mesothelioma according to death certificate information (out of 1,392 deaths, equivalent to 1%) compared to only one case in Charleston (out of 857 deaths, equivalent to 0.1%).<sup>98</sup> Moreover, the authors have raised the possibility that additional cases of peritoneal mesothelioma may have gone unrecognized and undiagnosed in the cohort.<sup>99</sup> We observe in any event that the authors have attributed the incidence of mesothelioma in this cohort to amphibole exposure, and predominantly to

<sup>94</sup> A.D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture."

<sup>95</sup> *Ibid.*, pp. 368-370 and Table 3, p. 370.

<sup>96</sup> *Ibid.*, Table 5, p. 371.

<sup>97</sup> The SMRs in the Pennsylvania plant were somewhat depressed relative to the SMRs at Charleston. The authors have tentatively suggested that a possible reason was that the employees had smoked fewer cigarettes than the general population of the state, perhaps because of the high proportion of Mennonites in the local community. However, no firm conclusions could be drawn.

<sup>98</sup> A.D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," pp. 372-373.

<sup>99</sup> *Ibid.*, p. 372. The authors noted that of the 14 mesothelioma deaths, 10 were pleural and 4 peritoneal. All 14 deaths occurred in the period from 1960 to 1975. Another 30 deaths, 15 or more years after first employment, were given the ICD Code 199 (malignant neoplasms of other and unspecified sites). Seventeen of these 30 deaths occurred before 1965, the year after which the majority of the mesothelioma deaths occurred. The diagnosis given in many of the cases did not preclude the possibility of an unrecognized peritoneal mesothelioma.

Table 5.10  
Male Deaths 20 Years After First Employment, by Cause, in Relation to Duration of Service

Cause of Death*	Length of Gross Service (Years)								Complete Cohort	
	<1		1<5		5<20		≥20			
	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR
All causes	171	87.2	154	106.2	187	104.5	383	127.2	895	109.0
Malignant neoplasms										
Respiratory	9	69.6	3	32.9	14	128.8	27	158.9	53	105.0
Abdominal	8	72.9	11	133.7	11	105.9	24	131.3	54	112.7
Other	19	132.4	16	152.0	15	118.5	32	155.3	82	141.1
Heart disease	77	92.7	77	125.1	78	100.2	153	115.7	385	108.5
Respiratory tuberculosis	0	—	1	133.4	0	—	2	67.3	3	51.7
Other respiratory	4	54.2	2	38.1	11	161.0	50	442.4	67	215.0
Pneumoconiosis	(2)	—	(1)	—	(10)	—	(46)	—	(59)	—
Cerebrovascular	7	54.9	10	106.5	10	77.7	20	87.6	47	81.2
Accidents	13	117.5	15	181.1	8	87.2	9	60.1	45	103.5
Other known	30	75.2	15	52.4	37	103.0	62	103.1	144	87.2
Not known	4		4		3		4		15	

**Note:** \*ICD Codes, except that ICD Codes 160-164 are here grouped under "respiratory" malignant neoplasms and the "other respiratory" category includes only bronchitis, pneumonia, and pneumoconiosis (ICD 490-502, 523-524).

SOURCE: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," *British Journal of Industrial Medicine* 40 (1983): 371 (Table 4).

amosite, although there was no specific exposure information reported on the individual cases.<sup>100</sup>

The mortality experience at this Pennsylvania plant has also been investigated by NIOSH.<sup>101</sup> Although the cohort was somewhat smaller (comprising 3,276 workers, excluding clerical employees, employed one year or more between 1940 and 1967), the mortality results for asbestos-related disease were consistent with those found by Dr. A.D. McDonald and her colleagues. Table 5.11 shows the observed and expected deaths, according to cause, among the 2,722 white male employees in the NIOSH study cohort. The overall SMR for lung cancer among the male employees was 136. There were 17 mesotheliomas recorded in the entire cohort, 13 among male employees and 4 among female employees.<sup>102</sup> A noteworthy finding of the study is the high rate of suicide. The major difference between the NIOSH study and that of Dr. A.D. McDonald and her colleagues relates to the amount of amphibole asbestos each reported as being used at the plant. As we have indicated, Dr. A.D. McDonald, on the basis of her research, concluded that crocidolite and amosite were used quite extensively.<sup>103</sup> NIOSH, on the other hand, claimed that in all but three years during World War II, chrysotile constituted over 99% of the asbestos processed; and while in those three years amosite was used for naval specifications, it still only accounted for 5% of asbestos consumption at the plant.<sup>104</sup> We are in no position to resolve this difference, but we do observe that in none of the 17 mesothelioma cases reported in the NIOSH study have the authors been able to isolate exposure to chrysotile alone.

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<sup>100</sup>Ibid., p. 373.

<sup>101</sup>See Joseph K. Wagoner, William M. Johnson, and Richard A. Lemen, "Malignant and Nonmalignant Respiratory Disease Mortality Patterns Among Asbestos Production Workers," in U.S. Congress, Senate, *Congressional Record*, 93rd Cong., 1st sess., 14 March 1973, vol. 119, pt. 6, pp. 7828-7830. This study was updated by Cynthia F. Robinson, Richard A. Lemen, and Joseph K. Wagoner, "Mortality Patterns, 1940-1975 Among Workers Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility," in *Dusts and Disease*, eds. Richard A. Lemen and John M. Dement (Park Forest South, Illinois: Pathatox Publishers, Inc., 1979), pp. 131-143.

<sup>102</sup>Robinson, Lemen, and Wagoner, "Mortality Patterns, 1940-1975 Among Workers Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility," pp. 138-139.

<sup>103</sup>Dr. A.D. McDonald and her colleagues indicated that crocidolite and amosite were used at the plant from 1924 onwards. They noted that much of the crocidolite was imported as yarn and only about 3 to 5 tons of raw fibre were used annually, but that amosite was extensively used, reaching a peak of 600 tons in 1943. According to the authors, chrysotile usage varied between 3,000 and 6,000 tons annually. See A.D. McDonald, "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," p. 369.

<sup>104</sup>Robinson, Lemen, and Wagoner, "Mortality Patterns, 1940-1975 Among Workers Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility," p. 133.

Table 5.11

**The Observed and Expected Deaths, According to Cause,  
Among White Males Employed in an Asbestos Textile,  
Friction and Packing Products Manufacturing Facility**

Cause of Death	List Number*	Observed	Expected	SMR
Malignant neoplasms	140-205	168	128.7	131***
Digestive system	150-159	50	41.4	121
Bronchogenic cancer	162-163	49	36.1	136**
Other and unspecified	140-149, 156B, 161, 164, 165, 170-199, 200-205	69	51.2	135**
Vascular lesions affecting the central nervous system	330-334, 345	49	57.0	86
Diseases of the heart	400-443	375	315.4	119**
Non-malignant respiratory disease	470-527	92	38.0	242***
Influenza, pneumonia, bronchitis, and acute upper respiratory infection	470-475, 480-483, 490-493, 500-502	16	21.4	74
Other respiratory disease	510-527	76	16.4	463***
Suicides	963, 970-979	30	17.1	175***
Other violent deaths	800-962, 964, 980-985	45	59.3	76
All other known causes		138	125.8	110
Unknown causes		15	0	—
Total		912	741.3	123**

**Notes:** \*Seventh Revision of the International Lists of Diseases and Causes of Death [now International Classification of Diseases].

\*\*Significant at  $p < 0.05$ .

\*\*\*Significant at  $p < 0.01$ .

SOURCE: Cynthia F. Robinson, Richard A. Lemen, and Joseph K. Wagoner, "Mortality Patterns, 1940-1975 Among Workers Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility," in *Dusts and Disease*, eds. Richard A. Lemen and John M. Dement (Park Forest South, Illinois: Pathatox Publishers, Inc., 1979), Table 2, p. 135.



## B.4 Asbestos Manufacturing — Cement Products

### (a) New Orleans, Louisiana

Dr. Hans Weill and his colleagues at Tulane University have now reported on the mortality experience of workers in two asbestos-cement building materials plants in New Orleans.<sup>105</sup> Chrysotile was the principal fibre used in both plants, but crocidolite was also used in the pipe department of the second plant where it constituted 3% of the final product. Dr. Weill has reported that the total percentage of asbestos fibre in most asbestos-cement products ranges from 15 to 28%,<sup>106</sup> so that crocidolite constituted between 10 and 20% of the asbestos used to make cement pipe.

As originally identified, the cohort studied by Dr. Weill included all workers employed in either of the two plants for at least one month prior to January 1, 1970. In order to maximize the likelihood of detecting adverse health effects, results were reported for the 5,645 men with a minimum follow-up through 1974 of 20 years from first employment.

Using social security records, Dr. Weill was only able to trace 75% of the cohort, 11% of whom had died. Dr. Weill's study has been criticized for its relatively poor trace rate, and it would appear that with the vital status of one-quarter of the cohort unknown, the possibility for bias in the results exists.<sup>107</sup> Moreover, as Dr. Weill assumed that all those not known to be dead were alive, it is quite possible that the disease risk has been underestimated in his study. On the other hand, Dr. Weill's trace rate was best among those most heavily exposed and where one would expect excess disease to be greater. Further, as Dr. Weill has pointed out, there is no necessity to assume any relationship between cause-specific mortality and trace rate.<sup>108</sup> In any event, Dr. Weill is currently updating his study and apparently has been able to trace a much larger percentage of his cohort.<sup>109</sup> Unfortunately, his updated results were not available in published form at the time this Report was being written.

In terms of the published study, no excess mortality occurred in any exposure group (measured in dust particles) for any cause other than lung

<sup>105</sup>Hans Weill, Janet Hughes, and Carmel Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease* 120:2 (August 1979): 345-354.

<sup>106</sup>*Ibid.*, p. 346.

<sup>107</sup>See, for example, RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 65; and Geoffrey Berry, "Discussion Summary," in *Biological Effects of Mineral Fibres*, vol. 2, p. 862.

<sup>108</sup>RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 68-70.

<sup>109</sup>Letter from Dr. Hans Weill, Pulmonary Diseases Section, Department of Medicine, Tulane Medical Center, New Orleans, Louisiana to the Royal Commission on Asbestos, 11 April 1983.

cancer. In turn, these rates showed excess risk only at exposure levels over 100 mppcf-yrs. Table 5.12 indicates standard mortality ratios, by cause of death, within various dust exposure categories.

The overall lung cancer mortality experienced by persons employed 10 or more years exhibited an SMR of 215 which, as Dr. Weill and his colleagues have pointed out, is remarkably similar to the corresponding figure of 217 found by Mr. Peto in his earlier 1978 mortality study at Rochdale.<sup>110</sup> There were only 2 mesotheliomas (both pleural) reported in the study, one employed only 10 months with known exposure only to chrysotile and the other employed for 14 years in the pipe plant thereby having exposure to both chrysotile and crocidolite.<sup>111</sup> However, Dr. Weill has recently advised us that more cases of mesothelioma have begun to appear in this cohort.<sup>112</sup>

In the published study, Dr. Weill separately analyzed the risk of lung cancer by fibre type. Among the 4,201 workers not exposed to crocidolite the SMR for lung cancer was only 77 (30 deaths versus 38.8 expected). Among the 235 maintenance workers intermittently exposed to crocidolite in the pipe plant the SMR was 304 (statistically significant at the 95% confidence interval), and among the 1,004 workers steadily employed in the pipe plant with crocidolite exposure the SMR was 155.<sup>113</sup> Leaving aside mesothelioma, the results of the Weill study appear to suggest that exposure to crocidolite and chrysotile in combination enhances the risk of lung cancer as compared to exposure to chrysotile alone, particularly for those workers exposed intermittently in maintenance jobs.

#### **(b) Manville Cement Workers**

In 1972–1973 and again in 1979, Dr. Philip E. Enterline and Ms. Vivian L. Henderson reported on the mortality experience of a group of men, aged 65 and over, who completed their working lifetimes as production or maintenance-service employees with the Johns-Manville

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<sup>110</sup>Weill, Hughes, and Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," p. 353.

<sup>111</sup>*Ibid.*, p. 349.

<sup>112</sup>Letter from Dr. Hans Weill to the Royal Commission on Asbestos, 11 April 1983.

<sup>113</sup>Weill, Hughes, and Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," pp. 351–352.

Table 5.12  
Standard Mortality Ratios, by Cause,  
Within Exposure Categories

Cause of Death	Total Dust Within 20 Years of Initial Exposure (mppcf-yrs)*									
	$\leq 10$		11-50		51-100		101-200		> 200	
	(n = 3,037)	SMR**	(n = 1,303)	O/E	(n = 383)	SMR	(n = 344)	O/E	(n = 578)	SMR
All causes	259/433.7	60	141/218.9	64	56/75.1	75	42/52.3	80	103/110.1	94
All malignant neoplasms (140-209)	54/77.3	70	27/37.1	73	7/12.9	54	14/9.5	147	18/19.8	91
Digestive system (150-159)	10/24.6	41	10/11.9	84	3/4.2	71	0/3.0	—	2/6.4	31
Respiratory system (160-163)	19/24.7	77	8/11.4	70	1/3.8	26	9/3.1	290†	14/6.2	226†
Other (residual)	25/28.0	89	9/13.8	65	3/4.9	61	5/3.4	147	2/7.2	28
Major cardiovascular diseases (390-448)	129/215.7	60	78/113.9	68	33/40.1	82	14/26.5	53	61/57.4	106
All other causes	76/140.7	54	26/47.8	54	12/16.2	74	9/11.6	78	20/23.8	84

**Notes:** \* Million particles per cubic foot-years.

\*\* Ratio of observed to expected deaths.

\*\*\* Standard mortality ratio.

† Significant at  $p < 0.01$  (number of observed deaths compared to the number expected, assuming a Poisson distribution).

SOURCE: Hans Weill, Janet Hughes, and Carmel Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease* 120:2 (August 1979): 348 (Table 2).

Corporation, who retired during the years 1941–1967, and who were exposed to asbestos dust in the course of their employment.<sup>114</sup> In their earlier report, the authors followed a total cohort of 1,464 retirees through to the end of 1969, by which time 822 had died. Of this cohort, 316 men who worked in the manufacture of cement shingles and sheets using only chrysotile asbestos had an overall lung cancer SMR of 142.9. A total of 106 retirees employed in the manufacture of asbestos-cement pipe using a combination of chrysotile and crocidolite had a lung cancer SMR of 588.2.<sup>115</sup> As this earlier investigation by Enterline and Henderson included Canadian employees, the asbestos-cement pipe workers undoubtedly included those retirees from the Johns-Manville plant at Scarborough, Ontario, who met the eligibility requirements to be part of the cohort.

In their more recent report, the authors excluded Canadian employees and thus had a cohort of 1,075 retirees averaging 25 years' employment, followed through to the end of 1973, by which time 781 men had died. Of this cohort, 402 retirees were identified as having been employed in the asbestos-cement industry: 305 in the production of rigid shingles and sheets using only chrysotile asbestos and 97 in the production of asbestos-cement pipe using both crocidolite and chrysotile asbestos.<sup>116</sup> Exposure levels were estimated on the basis of hygiene surveys, discussions with long-service employees, and a judgement as to the effect of changes in conditions. For the entire cohort, the SMR for lung cancer was 270.4.<sup>117</sup> While there were many Manville employees who died of mesothelioma prior to age 65, only 5 qualified for inclusion in the cohort, having died after age 65.<sup>118</sup>

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<sup>114</sup>Philip E. Enterline, Pierre DeCoufle, and Vivian L. Henderson, "Mortality in Relation to Occupational Exposure in the Asbestos Industry," *Journal of Occupational Medicine* 14:12 (December 1972): 897–903; Philip E. Enterline, Pierre DeCoufle, and Vivian L. Henderson, "Respiratory Cancer in Relation to Occupational Exposures Among Retired Asbestos Workers," *British Journal of Industrial Medicine* 30:1 (January 1973): 162–166; Philip E. Enterline and Vivian L. Henderson, "Type of Asbestos and Respiratory Cancer in the Asbestos Industry," *Archives of Environmental Health* 27:5 (November 1973): 312–317; and Vivian L. Henderson and Philip E. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 117–126.

<sup>115</sup>Enterline and Henderson, "Type of Asbestos and Respiratory Cancer in the Asbestos Industry," p. 316.

<sup>116</sup>Henderson and Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," p. 121.

<sup>117</sup>*Ibid.*, Table 1, p. 118. The all-cause SMR was 120.4, and the SMR for cancer of the digestive tract was 137.8. There were 31 observed cases of pneumoconiosis and pulmonary fibrosis in the cohort, of which 19 were due to asbestosis.

<sup>118</sup>*Ibid.*, pp. 124–125. The authors referred to a report by M. Borow et al. from hospitals in the vicinity of one of the plants studied by Henderson and Enterline, that of Manville, New Jersey. Borow reported 72 cases of mesothelioma from hospitals in the vicinity of this plant. A substantial number of these cases died prior to age 65, leading Henderson and Enterline to suggest that a feature in malignant mesothelioma is that death occurs at a relatively early age. See also, M. Borow et al., "Mesothelioma Following Exposure to Asbestos: A Review of 72 Cases," *Chest* 64 (November 1973): 641–646.



Focusing on the cement workers, for those working with chrysotile alone the SMR for lung cancer was 230.8 with a mean exposure of 255 mppcf-yrs; for those working with crocidolite and chrysotile in the cement pipe operations the SMR for lung cancer was very much higher at 521.7 with a mean exposure of 230 mppcf-yrs. There were 11 deaths from asbestosis among the cement shingle and sheet workers and only 2 among the cement pipe workers.<sup>119</sup>

### (c) Scarborough, Ontario

We have already described in general terms, in Chapter 3, the disastrous health experience of the asbestos-cement workers at the Johns-Manville plant at Scarborough, Ontario, as reported by Dr. Murray M. Finkelstein. It will be recalled that this factory began production in 1948 manufacturing asbestos-cement pipe in one building and rock wool insulation in another. In 1955, production of asbestos-cement board commenced in a third building, and in 1960, the manufacture of asbestos insulation material was added.<sup>120</sup> The manufacture of asbestos-cement pipe involved both chrysotile and crocidolite, with crocidolite constituting approximately 20% of the asbestos used. As at New Orleans, it would appear that crocidolite constituted approximately 3% of the final product. In the manufacture of asbestos-cement board only chrysotile was used and this product was no longer made after 1970. No asbestos was used in the manufacture of rock wool insulation and these workers served as an internal control group.

Exposure information from the plant was scanty. Ontario Ministry of Labour hygienists performed surveys in 1949, 1956, 1961, 1968, and during the 1970s. Impinger surveys were performed for Johns-Manville by insurance company hygienists in 1954 and 1957, and Johns-Manville itself began annual measurements in 1961. However, there were no fibre counts until 1969, and reliable membrane filter sampling results from the government were not available until 1973.<sup>121</sup> There were two major ventilation changes in the pipe plant, the first, according to Dr. Finkelstein, having taken place in 1962 and the second in 1970.<sup>122</sup>

Based on the available data, Dr. Finkelstein made the following assumptions: (i) the relative dustiness of the jobs was unchanged between

<sup>119</sup>Henderson and Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," pp. 121-123.

<sup>120</sup>See Chapter 3, Section A.3 of this Report.

<sup>121</sup>Murray M. Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," *American Review of Respiratory Disease* 125 (1982): 497. See also, RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 46-48.

<sup>122</sup>Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 497.

1948 and 1970; (ii) between 1955 and 1962, exposures were 30% higher than those in 1969–1970; (iii) exposures between 1948 and 1954 were twice as high as exposures in 1969–1970; and (iv) from 1962 to 1970, exposures were unchanged. Dr. Finkelstein constructed an exposure-response model using these assumptions in conjunction with the pre-ventilation change 1969–1970 fibre counts.<sup>123</sup>

We have already observed that Dr. Finkelstein conducted two separate analyses of the mortality experience of these workers: the first restricted to those 339 workers hired prior to 1960 and with at least 9 years' employment followed to October 1980;<sup>124</sup> the second extended to a cohort of 740 men employed one year or more, hired prior to 1960 and followed to the end of 1981.<sup>125</sup>

<sup>123</sup> Ibid. See also, Murray M. Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," *British Journal of Industrial Medicine* 40 (1983): 139.

<sup>124</sup> Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 139. Of the 339 workers, 11 could not be classified from their work histories and were excluded from the analysis. Of the remaining 328 workers, 186 were production workers, 55 were maintenance workers, and 87 were employed in the rock wool/fibreglass operations or were otherwise minimally exposed to asbestos dust and thus constituted the control group.

<sup>125</sup> Murray M. Finkelstein, "Mortality in Asbestos-Cement Factory Workers," paper presented at the Second International Symposium on Epidemiology in Occupational Health, Montreal, 25 August 1982, pp. 1–2. (Mimeographed.) See also, Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983. (Mimeographed.) The composition of the cohort was as follows:

**Numbers of Men at Risk in the Cohort  
and the Numbers Withdrawn from the Analysis  
at the End of 1977**

	<u>Years From Hire</u>		<u>Number Withdrawn</u>	<u>Percent Withdrawn</u>
	<u>10</u>	<u>20</u>		
Production Workers	428	401	63	16%
Maintenance Workers	107	99	13	13%
Factory Controls	205	185	32	17%
Total	740	685	108	16%

SOURCE: Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983, Table 1, excerpt. (Mimeographed.)

The difference in numbers at the two dates from hire is due to death or emigration in the interval. All production workers were exposed to asbestos for at least one month prior to the end of 1961. Exposure-response calculations were made for the 401 production workers alive 20 or more years from their date of hire.

In the more restricted cohort of longer-term employees, among those with an average 18 years' cumulative exposure roughly estimated to be 180 f/cc-yrs, he observed a mesothelioma mortality rate of approximately 12 per thousand and an equal rate for lung cancer.<sup>126</sup> Even setting aside the exposure estimates, a striking statistic from this cohort is that, of the 58 deaths among production workers, 20 were caused by lung cancer and 10 more by mesothelioma. Table 5.13 shows mortality among the longer-term Scarborough factory workers, by selected causes of death, compared with the mortality predicted from Ontario population rates.

In Dr. Finkelstein's larger cohort of workers employed one year or more, the disease incidence has remained extraordinarily high. An indication of this fact can be gained from the mortality data for production workers 20 or more years from first employment: the SMR for all causes of death was 181; for lung cancer, 512; for gastrointestinal cancer, 285; and for non-malignant respiratory disease, 320 — figures which are clearly of the order of magnitude found by Dr. Dement and Dr. A.D. McDonald at Charleston, but which are considerably higher than those reported by Dr. Weill in New Orleans. Table 5.14 details observed and expected mortality for Dr. Finkelstein's larger cohort.

Dr. Finkelstein has also calculated 18-year cumulative exposures for the production workers in his extended cohort by combining work histories with the exposure estimates. Table 5.15 shows standardized mortality rates for these workers, for the period 20 or more years from first exposure, in relation to various exposure categories.

Of the 21 mesothelioma deaths which had already occurred in this cohort, 12 were pleural and 9 peritoneal, and it would appear that all but one had exposure to crocidolite in the pipe plant. Mesothelioma mortality rates increased significantly with time from first exposure.<sup>127</sup>

Dr. Finkelstein's study has been criticized for the uncertainty of the exposure information and for the fact that a clear dose-response relationship was not demonstrated for lung cancer. Dr. Finkelstein, in his testimony before the Commission, freely recognized these difficulties, but, as he also pointed out, his data do not compare unfavourably with those available in

<sup>126</sup>Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 139. The mortality rate for lung cancer for Ontario men per 1,000 man-years is 1.6.

<sup>127</sup>Finkelstein, "Mortality in Asbestos-Cement Factory Workers," pp. 4-5. In this paper, Dr. Finkelstein indicated that 2 of the mesotheliomas occurred in the control group, but that one of these men had later moved to the Transite pipe section of the plant. The other man was not recorded as having worked in the asbestos areas. However, Dr. Finkelstein did observe that it was not uncommon for men from other areas of the plant to do weekend cleanups in the pipe shop, and these men may have been exposed in that fashion.

Table 5.13  
Mortality Among the Factory Workers Compared with the Population of Ontario

Cause	Group*	Years Since First Exposure								
		15-19			20-24			25-33		
		Obs	Exp	O/E	Obs	Exp	O/E	Obs	Exp	O/E
All causes	P	8	8.9	1	16	11.8	1.4	34	11.6	2.9
	P + M	11	11.9	1	22	15.5	1.4	39	15.1	2.6
	C	7	5.0	1.4	7	5.9	1.2	7	7.4	1
All malignancies ICD: 140-209	P	2	1.9	1	9	2.8	3.2	20	2.9	6.9
	P + M	2	2.5	1	11	3.7	3.0	23	3.7	6.2
	C	3	1.1	2.7	3	1.4	2.1	1	1.8	1
Lung cancer ICD: 162	P	1	0.6	1	6	1.0	6.0	11	1.0	11.0
	P + M	1	0.8	1	7	1.2	5.8	12	1.3	9.2
	C	0	0.3	0	0	0.5	0	1	0.6	1
Mesothelioma ICD: 163, 158, 228	P	1	—	—	2	—	—	4	—	—
	P + M	0	0.5	0	1	0.7	1	2	0.7	2.9
	C	1	0.3	1	1	0.3	1	0	0.4	0
Non-malignant respiratory disease ICD: 460-519	P	1	0.4	1	1	0.7	1	3	0.8	3.8
	P + M	1	0.6	1	3	0.9	3.3	4	1.0	4.0
	C	0	0.3	0	0	0.4	0	1	0.5	1
Ischemic heart disease ICD: 410-414	P	4	3.9	1	2	4.7	0.4	5	4.6	1
	P + M	7	4.9	1.4	3	6.2	0.5	6	6.0	1
	C	3	2.1	1	1	2.4	0.4	2	2.9	1
Total: 20-33		Obs	Exp	O/E	Obs	Exp	O/E	Obs	Exp	O/E
		50	23.4	2.1	34	11.6	2.9	34	23.4	2.1
		61	30.6	2.0	39	15.1	2.6	61	30.6	2.0
		14	13.3	1	7	7.4	1	14	13.3	1
		29	5.7	5.1	20	2.9	6.9	29	5.7	5.1
		34	7.4	4.6	23	3.7	6.2	34	7.4	4.6
		4	3.2	1	1	1.8	1	4	3.2	1
		17	2.0	8.5	11	1.0	11.0	17	2.0	8.5
		19	2.5	7.6	12	1.3	9.2	19	2.5	7.6
		1	1.1	1	1	0.6	1	1	1.1	1
		6	—	—	4	—	—	6	—	—
		3	1.4	2.1	2	0.7	2.9	3	1.4	2.1
		4	1.8	2.2	3	0.9	3.3	4	1.8	2.2
		1	0.7	1	0	0.4	0	1	0.7	1
		4	1.5	2.7	3	0.8	3.8	4	1.5	2.7
		7	1.9	3.7	4	1.0	4.0	7	1.9	3.7
		1	0.9	1	1	0.5	1	1	0.9	1
		7	9.3	0.8	5	4.6	1	7	9.3	0.8
		9	12.2	0.7	6	6.0	1	9	12.2	0.7
		3	5.3	0.6	2	2.9	1	3	5.3	0.6

Notes: \*P means production workers. M means maintenance workers. C means factory controls (unexposed workers).

SOURCE: Murray M. Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," *British Journal of Industrial Medicine* 40 (1983): 140 (Table 2).



Table 5.14  
Mortality Among the Factory Workers Compared  
with the General Population of Ontario

		Years Since First Exposure											
		10 - 14		15 - 19		20 - 24		25 - 29		30 - 34		Total: 20 - 34	
Cause	Group*	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR
All causes	P	6	50	17	96	26	113	50	272	10	161	86	181
	M	2	48	5	84	9	134	10	164	3	163	22	150
	C	4	56	12	123	12	102	9	78	7	130	28	98
All malignancies	P	0(1)**	[2.2]**	3(4)	79	14(17)	255	25(28)	543	5	333	44(50)	379
ICD: 140-209	M	1	125	1	79	2	124	7	461	3	638	12	333
(8th Revision)	C	1(2)	71	3	144	4(6)	191	2	71	3	234	9(11)	131
Lung cancer	P	0	[0.6]	2(3)	167	7(7)	368	13(13)	813	1	167	21(21)	512
ICD: 162	M	1	476	1	263	1	182	2	370	2	1,180	5	413
	C	0(1)	[0.4]	0	[0.7]	0(1)	[0.9]	1	103	2(1)	476	3(3)	130
Mesothelioma	P	0(1)		1		2(5)		4(7)		2(3)		8(15)	
ICD: 228, 158, 163	M	0		0		1		1		0		2(2)	
	C	0		0		0		0(1)		0(1)		0(2)	
$\left. \begin{array}{l} \text{ } \\ \text{ } \end{array} \right\} = 0.4 \text{ per } 10^3$ $\left. \begin{array}{l} \text{ } \\ \text{ } \end{array} \right\} = 2.7 \text{ per } 10^3$ $\left. \begin{array}{l} \text{ } \\ \text{ } \end{array} \right\} = 6.3 \text{ per } 10^3$ $\left. \begin{array}{l} \text{ } \\ \text{ } \end{array} \right\} = 9.6 \text{ per } 10^3$ $\left. \begin{array}{l} \text{ } \\ \text{ } \end{array} \right\} = 4.4 \text{ per } 10^3$													
Gastrointestinal	P	0	[0.6]	0	[0.9]	3(2)	231	3(5)	273	2(1)	500	8(8)	285
cancer	M	0	[0.2]	0	[0.3]	0	[0.4]	1	278	0	[0.1]	1	118
ICD: 150-154	C	0	[0.4]	1	192	0(1)	[0.7]	1(0)	152	0	[0.3]	1(1)	61

Table 5.14 (continued)  
Mortality Among the Factory Workers Compared  
with the General Population of Ontario

		Years Since First Exposure										Total: 20 - 34	
Cause	Group*	10 - 14		15 - 19		20 - 24		25 - 29		30 - 34		O	SMR
Non-malignant respiratory diseases ICD: 460-519	P	0	[0.4]	1	122	2(1)	154	7(6)	636	0(1)	[0.4]	9(8)	320
	M	0	[0.2]	0	[0.3]	3	789	1	256	0	[0.1]	4	444
	C	0	[0.3]	0	[0.5]	0	[0.7]	1	130	0	[0.4]	1	54
Ischemic heart disease	P	4	86	7	98	2(1)	22	6	82	3	120	11(10)	58
	M	0	[1.7]	4	163	2	74	1	41	0	[0.7]	3	51
	C	1	35	4	100	5(4)	106	4	89	3	141	12(11)	106

**Notes:** \* P means production workers. M means maintenance workers. C means factory controls (unexposed workers).

\*\*Numbers in parentheses reflect "best evidence" classification.

\*\*\*Numbers in brackets are numbers of deaths expected.

SOURCE: Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983, Table 2. (Mimeographed.)

Table 5.15  
Mortality Rates in Relation to Cumulative Exposure

Cause of Death	Mortality Rates per 10 <sup>3</sup> Man-Years*							Test for Trend
	Ontario Males**	Factory Controls	≤ 30 f/cc-yrs	30.1-75 f/cc-yrs	75.1-105 f/cc-yrs	105.1-150 f/cc-yrs	> 150 f/cc-yrs	
Lung cancer	1.3	1.7 (3)†	3.0 (3)	8.0 (6)	15.7 (5)	11.7 (5)	3.5 (2)	Z = 2.5; P < 0.01.
Mesothelioma	≪ 1	1.1 (2)	4.1 (4)	3.8 (3)	2.4 (1)	2.7 (1)	17.9 (6)	Z = 3.5; P < 0.01.
Gastrointestinal cancer	1.0	0	2.0 (2)	3.5 (2)	2.4 (1)	0	10.1 (3)	Z = 2.3; P < 0.02.
All asbestos cancer	2.3	2.8 (5)	9.1 (9)	15.4 (11)	20.6 (7)	14.4 (6)	31.5 (11)	
Non-malignant respiratory disease	1.2	0.6 (1)	2.0 (2)	1.4 (1)	4.9 (2)	6.2 (2)	0.9 (1)	Z = 1.6; 0.05 < P < 0.06.
Ischemic heart disease	7.4***	6.4 (11)	1.0 (1)	1.5 (1)	6.7 (3)	17.8 (4)	2.4 (1)	Z = 0.3; P > 0.3.
All causes of death	17.9	16.2	20.1	26.7	40.2	39.6	41.4	Z = 4.4; P < 0.01.
Number of men in exposure category at 20 years	185	169	113	37	43	39		
Total number of deaths 20 - 34 years	28	20	21	15	13	17		

**Notes:** \*Standardized to the age and latency distribution of the cohort as a whole, for the period beyond 20 years from first exposure.

\*\*From Ontario Vital Statistics 1970-1974.

\*\*\*From Ontario Vital Statistics 1969-1973.

†Numbers in parentheses are the actual numbers of deaths.

SOURCE: Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983, Table 6. (Mimeographed.)

many other studies. Moreover, he estimates that his calculations of cumulative exposure are accurate to within a factor of 3 to 5.<sup>128</sup> Notwithstanding the potential for error arising out of the uncertainty of the exposure information, the disease experience at Scarborough is remarkably high.

#### (d) Cardiff, Wales

H.F. Thomas et al. of the Medical Research Council Epidemiology Unit at Cardiff, Wales, have recently reported on the mortality experience of 1,970 workers employed at an asbestos-cement factory near Cardiff for at least 6 months between 1936 and 1977.<sup>129</sup> Crocidolite was used at this factory between 1932 and 1935, but from 1935 onwards until the factory closed in 1980 only chrysotile from Canada and South Africa was used, principally for the manufacture of cement sheeting and associated fittings for the building industry. Over 95% of the cohort was traced by the end of 1977 and 381 deaths were identified.

Detailed analysis was restricted to the 1,592 male employees among whom there were 351 observed deaths. These male workers were separated into three categories: all male workers; those alive 15 or more years after first exposure; and those employed in 1935–1936 and thus potentially exposed to crocidolite. In none of the three groups was there any apparent increase in general mortality, cancer mortality, or in mortality specifically from lung cancer and gastrointestinal cancer. Table 5.16 shows the actual standard mortality ratios for selected causes of death. There were 2 cases of pleural mesothelioma in the cohort, and both were employed at the factory when crocidolite was used.<sup>130</sup> Thus, the general results of the study suggest that the chrysotile cement workers are not at any increased risk from asbestos exposure. This conclusion, however, must be treated with some caution. There was no minimum latency period for the entire cohort. Further, over half the cohort studied was employed less than 2 years, and three-quarters worked in the factory for less than 4 years. No individual exposure data have been presented for the cohort, although the authors commented that dust levels in asbestos-cement factories are generally lower than in other asbestos-handling industries, and in the Cardiff plant there was a pronounced improvement in dust levels from the mid-1960s onwards. The authors concluded their published study by commenting that while it “. . . gives reassuring findings on the long-term mortality experience of an

<sup>128</sup>The issue of Dr. Finkelstein's exposure estimate is discussed in greater detail in Chapter 3, Section A.3.

<sup>129</sup>H.F. Thomas et al., "Further Follow-up Study of Workers from an Asbestos Cement Factory," *British Journal of Industrial Medicine* 39:3 (August 1982): 273–276.

<sup>130</sup>*Ibid.*, pp. 274–275.



Table 5.16  
Mortality by All Causes, All Neoplasms, and Selected Cancers for the Total Male Population,  
Men Alive 15 Years or Longer After First Exposure, and for Men Employed in 1935-1936

Cause of Death (ICD Code)	Study Group	Expected Deaths	Observed Deaths	Standardized Mortality Ratio (SMR)
All causes	Total population	343.3	351	102
	Alive $\geq$ 15 years after first exposure	243.2	261	107
	Employed in 1935-1936	88.3	83	94
All neoplasms (140-239)	Total population	80.7	74	92
	Alive $\geq$ 15 years after first exposure	61.0	58	95
	Employed in 1935-1936	21.5	22	103
Cancers of lung and pleura (162, 163)	Total population	33.0	30	93
	Alive $\geq$ 15 years after first exposure	25.8	24	93
	Employed in 1935-1936	9.2	7	76
Cancers of gastrointestinal tract (151-154)	Total population	19.6	18	92
	Alive $\geq$ 15 years after first exposure	14.1	14	99
	Employed in 1935-1936	5.0	6	120

SOURCE: H.F. Thomas et al., "Further Follow-up Study of Workers from an Asbestos Cement Factory," *British Journal of Industrial Medicine* 39:3 (August 1982): 274 (Table 2).

asbestos cement factory population, the high labour turnover does not enable conclusions to be drawn on the hazards of sustained long-term exposure to chrysotile asbestos."<sup>131</sup>

## B.5 Asbestos Manufacturing — Amosite Insulation Products

### Paterson, New Jersey

Just prior to the entry of the United States into World War II, the Union Asbestos and Rubber Company (UNARCO) established an amosite asbestos factory at Paterson, New Jersey, to supply the U.S. Navy with asbestos insulation for the pipes and boilers of its ships. From June 1941 until December 1945, 933 men were hired to work in the plant which continued in operation until 1954. The health experience of these workers was reported on by Dr. Herbert Seidman and his colleagues at the Mount Sinai School of Medicine in New York in 1979.<sup>132</sup>

As the employees of Paterson were among those not conscripted for war service, they tended to be considerably older in age than a peace-time workforce. Most worked for short periods of time, many for less than 9 months, and only a small number worked longer than 2 years. Following the closing of the Paterson plant, few returned to employment with asbestos.<sup>133</sup> There were no direct measurements of dust levels in the plant; however, measurements made more recently in two other plants operated by UNARCO — one in Tyler, Texas, and the other in Port Allegany, Pennsylvania — making the same product, from the same fibre type (amosite), to the same specifications, and using the same type of machinery, were extremely high, averaging 34.9 f/cc.<sup>134</sup>

<sup>131</sup> Ibid., pp. 275–276.

<sup>132</sup> Herbert Seidman, Irving J. Selikoff, and E. Cuyler Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 61–89. There is an earlier report of the same cohort by Herbert Seidman, Ruth Lilis, and Irving J. Selikoff, "Short-Term Asbestos Exposure and Delayed Cancer Risk," in *Prevention and Detection of Cancer*, vol. 1: *Etiology*, ed. H.E. Nieburgs (New York: Marcel Dekker, Inc., 1977), pp. 943–960.

<sup>133</sup> Seidman, Selikoff, and Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," p. 62.

<sup>134</sup> In their published article, the authors suggested dust counts as high as 23 f/cc. The 34.9 f/cc figure comes from William J. Nicholson, "Criteria Document for Swedish Occupational Standards: Asbestos and Inorganic Fibers," *Arbete Och Hälsa* 17 (1981): 22–23. The figure was based upon measurements made by the U.S. Public Health Service during 1967, 1970, and 1971. The arithmetic averages of these exposure measurements for Tyler and for Port Allegany were 39.1 and 28.9 f/cc respectively, yielding an overall average of 34.9 f/cc.

The health experience at Paterson therefore reflects both the unusual characteristics of the workforce and the rather unusual nature of their asbestos exposure — short but very intense exposure (to amosite) — followed by a long period of observation, 35 or more years from first exposure. In order to investigate only the health effects of asbestos exposure at Paterson, Dr. Seidman excluded for the purpose of analysis 113 employees who had asbestos exposure elsewhere, leaving 820 in the cohort.

By the time he reported, 528 workers in the cohort had died, 93 from lung cancer and 14 from mesothelioma, and the workers employed 2 or more years were close to extinction (157 deaths out of 188 workers).<sup>135</sup> Lung cancer mortality rates were extremely high, with SMRs ranging from 294 for those with less than one month's employment to 650 for those employed more than 2 years.<sup>136</sup> Many were already at or near "cancer ages" when hired and died within a relatively short period of time, 5 to 14 years after onset of work.<sup>137</sup>

Based on the health experience at Paterson, which is admittedly marked by unusual circumstances, working with amosite asbestos in the manufacture of insulation products for just one year at 1 f/cc would yield an excess risk of lung cancer of 9.1%. Working for 10 years at 1 f/cc would double that risk.<sup>138</sup>

## B.6 Asbestos Outside Fixed Place Industry

### North American Insulators

Commencing in the 1960s, Dr. Irving J. Selikoff and his associates at the Mount Sinai School of Medicine in New York have been following the mortality experience of a large group of insulation workers in the United

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<sup>135</sup>Seidman, Selikoff, and Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," pp. 66–67.

<sup>136</sup>Nicholson, "Criteria Document for Swedish Occupational Standards: Asbestos and Inorganic Fibers," p. 21.

<sup>137</sup>Seidman, Selikoff, and Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," pp. 87–88.

<sup>138</sup>RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 65.

States and Canada.<sup>139</sup> These workers were members of the International Association of Heat and Frost Insulators and Asbestos Workers. They have been primarily employed in the building trades (engaged in doing construction insulation work), but have also worked in refineries, shipyards, and on repair jobs. These insulators have generally been active in all parts of the trade with conditions varying from job to job and from company to company. According to Dr. Selikoff, fewer than 10% have remained with one company during their working lives.<sup>140</sup>

Until approximately the early 1930s, chrysotile alone was utilized in the manufacture of the insulation products used by these men. Amosite began to be used in the mid-1930s in small quantities but became more extensively utilized during and following World War II. Few dust counts were made in insulation work until the 1960s, and Dr. Selikoff has presented no exposure data in his studies. He has, however, suggested, based on reconstruction of work situations and extrapolation to the past of observations made more recently, that these insulation workers would have been exposed to dust levels of between 4 and 12 f/cc. While there may have been work periods of little or no exposure, there were also likely times of peak exposures much higher than the calculated averages.<sup>141</sup>

Dr. Selikoff has conducted mortality analyses on two cohorts of insulators. The first was a small group of 632 workers who, as of January 1, 1943, were registered as members of the union in the New York - New Jersey metropolitan area. By the end of 1976, 478 of the original 632 workers had died. Excess mortality due to asbestos-related diseases was striking. Of the total deaths in the cohort, 93 were from lung cancer, 38 from mesothelioma (11 pleural and 27 peritoneal), and 41 from asbestosis (of which 38 were in the period 35 or more years from first exposure). The SMRs for lung cancer, cancer of the stomach, and cancer of the colon or rectum were respectively 699, 352, and 277.<sup>142</sup>

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<sup>139</sup>Irving J. Selikoff, Jacob Churg, and E. Cuyler Hammond, "Asbestos Exposure and Neoplasia," *Journal of the American Medical Association* 188 (1964): 22-26; Irving J. Selikoff, Jacob Churg, and E. Cuyler Hammond, "The Occurrence of Asbestosis Among Insulation Workers in the United States," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 139-155; Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Cancer Risk of Insulation Workers in the United States," in *Biological Effects of Asbestos*, eds. P. Bogovski et al., IARC Scientific Publications, no. 8 (Lyon, France: International Agency for Research on Cancer, 1973), pp. 209-216; and Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91-116.

<sup>140</sup>Selikoff, Hammond, and Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," pp. 91-92.

<sup>141</sup>*Ibid.*, pp. 92-93.

<sup>142</sup>*Ibid.*, pp. 93-95.



The second cohort investigated by Dr. Selikoff was very much larger and comprised all 17,800 workers on the union rolls both in Canada and the United States as of January 1, 1967. Observation of this group was maintained through to the end of 1976, and in that 9-year period there were 2,271 deaths. According to the best available evidence, 175 of these deaths were from mesothelioma (63 pleural and 112 peritoneal), 168 from asbestosis, and 486 from lung cancer. Altogether these three causes of death were responsible for approximately 37% of all deaths in the cohort. The SMRs for lung cancer, gastrointestinal cancer, and cancer of the larynx were respectively 460, 167, and 234. (See Table 5.17.) The authors of the study justifiably concluded that these asbestos insulation workers suffered an extraordinarily increased risk of death from cancer and from asbestosis associated with their employment. It should be noted that little of the excess mortality was observed in the period less than 15 years from first exposure and most deaths occurred two, three, and four or more decades from the onset of exposure.<sup>143</sup>

## B.7 Summary

This review of the epidemiological evidence, which is summarized in Table 5.18, has served to illustrate that there does appear to be an association between the incidence of asbestos-related disease and the type of industrial process in which asbestos is used. The health risk in chrysotile friction products manufacturing and chrysotile mining and milling pales by comparison to the health risk in chrysotile textile production. Insulators seem to be at greater risk than miners and millers and most manufacturing workers.

And yet, the differences in industrial processes do not account for all of the observed differences in the results of the various epidemiological studies. Even among plants using similar processes and making similar products there has been a wide divergence in the incidence of disease. The mortality experience of the asbestos-cement workers at Scarborough ranks on a par with that of the textile workers at Charleston and has been considerably worse than the experience of the New Orleans workforce. In turn, the cement workers in New Orleans and among Dr. Enterline's Manville employees have fared worse than the chrysotile cement workers at Cardiff. The overall incidence of asbestos-related disease at Rochdale, while not insignificant and roughly similar to that at Lancaster, has been less than that experienced at Charleston. The incidence of mesothelioma in the mines of Western Australia and of the Cape region in South Africa has been markedly different than its incidence in the Quebec mines or even apparently in the mining region of the Transvaal. The number of mesotheliomas at

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<sup>143</sup> Ibid., pp. 106-110.

Table 5.17

**Deaths Among 17,800 Asbestos Insulation Workers in the United States  
and Canada, January 1, 1967 - December 31, 1976**  
(Number of Men: 17,800; Man-Years of  
Observation: 166,853)

Underlying Cause of Death	Expected*	Observed		Ratio O/E	
		(BE)	(DC)	(BE)	(DC)
Total deaths, all causes	1,658.9	2,271	2,271	1.37	1.37
Total cancer, all sites	319.7	995	922	3.11	2.88
Cancer of lung	105.6	486	429	4.60	4.06
Pleural mesothelioma	**	63	25	—	—
Peritoneal mesothelioma	**	112	24	—	—
Mesothelioma, not otherwise stated	**	0	55	—	—
Cancer of esophagus	7.1	18	18	2.53	2.53
Cancer of stomach	14.2	22	18	1.54	1.26
Cancer of colon-rectum	38.1	59	58	1.55	1.52
Cancer of larynx	4.7	11	9	2.34	1.91
Cancer of pharynx, buccal	10.1	21	16	2.08	1.59
Cancer of kidney	8.1	19	18	2.36	2.23
All other cancer	131.8	184	252	1.40	1.91
Non-infectious pulmonary diseases, total	59.0	212	188	3.59	3.19
Asbestosis	**	168	78	—	—
All other causes	1,280.2	1,064	1,161	0.83	0.91

**Notes:** \*Expected deaths are based upon white male age-specific U.S. death rates of the U.S. National Center for Health Statistics, 1967-1976.

\*\*Rates are not available, but these have been rare causes of death in the general population.

(BE): Best evidence. Number of deaths categorized after review of best available information (autopsy, surgical, clinical).

(DC): Number of deaths as recorded from death certificate information only.

SOURCE: Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 103 (Table 12).

**Table 5.18**  
**Mortality from Asbestos-Related Diseases in Various Cohort Studies**

Type of Activity	Study	Place	Fibre Type
Mining	J.C. McDonald, 1980	Quebec	Chrysotile
	Nicholson, 1979	Quebec	Chrysotile
	Rubino, 1979	Italy	Chrysotile
	Hobbs, 1980	Western Australia	Crocidolite
	Meurman, 1974	Finland	Anthophyllite
Friction Materials	Berry, 1983	England	Chrysotile, Crocidolite <sup>m</sup>
	A.D. McDonald, 1984	Connecticut	Chrysotile, Crocidolite <sup>m</sup> , Anthophyllite <sup>m</sup>
Textiles	Peto, 1977	England	Chrysotile, Crocidolite (?)
	Peto, 1980	England	Chrysotile, Crocidolite (?)
	Dement, 1982	South Carolina	Chrysotile, Crocidolite <sup>m</sup>
	A.D. McDonald, 1983	South Carolina	Chrysotile, Crocidolite <sup>m</sup>
	A.D. McDonald, 1983	Pennsylvania	Chrysotile, Amosite, Crocidolite <sup>m</sup>
	Robinson, 1979	Pennsylvania	Chrysotile, Amosite <sup>m</sup> , Crocidolite <sup>m</sup>
Cement Products	Weill, 1979	New Orleans	Chrysotile, Crocidolite, Amosite <sup>m</sup>
	Finkelstein, 1983	Scarborough	Chrysotile, Crocidolite
	Thomas, 1982	Cardiff	Chrysotile, Crocidolite <sup>m</sup>
General Manufacturing	Henderson and Enterline, 1979	United States	Chrysotile, Crocidolite, Amosite
	Newhouse, 1979	England	Chrysotile, Crocidolite, Amosite
Gas Mask Manufacturing	Jones, 1980	England	Crocidolite
Insulation Products	Seidman, 1979	New Jersey	Amosite
Insulators	Newhouse, 1979	England	Chrysotile, Crocidolite, Amosite
	Selikoff, 1979	United States and Canada	Chrysotile, Amosite
	Selikoff, 1979	New York — New Jersey	Chrysotile, Amosite
Shipyard Workers	Rossiter, 1980	England	Chrysotile, Crocidolite, Amosite

	Number in Cohort	Total Deaths	Meso-thelioma	Asbestosis (Pneumo-coniosis)	Lung Cancer			Gastrointestinal Cancer <sup>a</sup>		
					Obs	Exp	SMR	Obs	Exp	SMR
	10,939	3,291	10	42	230	184	125	209	203	103
	544	178	1	26	28	11.1	252	10	9.5	105
	952	332	0	9	11 <sup>b</sup>	10.4	106	19	19.3	98
	6,200	526	17 <sup>c</sup>	14	60	38.2	157	NS		
	1,092	248	0	13	21	12.6	167	7	14.9	47
M	9,113	1,640	8	NS	143 <sup>d</sup>	139.5	103	103	107.2	96
F	4,347	346	2	NS	6 <sup>d</sup>	11.3	53	29	27.4	106
	3,641	1,267	0	12 <sup>e</sup>	73 <sup>f</sup>	49.1	148.7	59 <sup>f</sup>	51.6	114.4
	1,106	317	10	NS	51 <sup>d</sup>	23.8	214	16	15.7	102
	679	239	7	10	40	23.3	172	NS		
	768	191	1	15	26	7.5	348	9	7.1	127
	2,543	857	1	21	59 <sup>f</sup>	29.6	199.5	26 <sup>f</sup>	17.1	151.7
	4,137	1,392	14	74	53 <sup>f</sup>	50.5	105	54 <sup>f</sup>	47.9	112.7
M	2,722	912	13	NS	49	36.1	136	50	41.4	121
F	554	128	4	NS	14	1.7	824	8	6.0	133
	5,645	601	0 <sup>g</sup>	NS	51	49.2	104	25	50.1	50
	535	138	19	NS	26 <sup>f</sup>	5.4	480	9 <sup>f</sup>	3.8	240
	1,592	351	2	NS	28	33.0	85	18	19.6	92
	1,075	781	5	31	63 <sup>h</sup>	23.3	270.4	55	39.9	137.8
M	2,887	545	46	NS	103 <sup>d</sup>	43.2	238	40 <sup>i</sup>	34	118
F	693	200	21	NS	27 <sup>d</sup>	3.2	844	20 <sup>i</sup>	10.2	196
	578	166	17	NS	12	6.3	190	10	20.3	49
	820	528	14 <sup>i</sup>	30 <sup>j</sup>	93 <sup>i</sup>	22.8	408	NS		
	1,368	83	10	NS	21 <sup>d</sup>	5.6	375	3 <sup>i</sup>	4.3	70
	17,800	2,271	175 <sup>j</sup>	168 <sup>j</sup>	486 <sup>j</sup>	105.6	460	99 <sup>j</sup>	59.4	167
	632	478	38 <sup>j</sup>	41 <sup>j</sup>	93	13.3	699	43	15.1	285
	6,076	1,043	31	9	84	119.7	70	63	83.3	76



Table 5.18 (continued)

## Mortality from Asbestos-Related Diseases in Various Cohort Studies

<b>Notes:</b>	<p><sup>a</sup>The figures in this table for "Gastrointestinal Cancer" may not be entirely comparable among the various cohort studies since some studies give figures for all abdominal or digestive cancers and others for gastrointestinal cancer only. The figures used in the actual studies have been reproduced in this table.</p> <p><sup>b</sup>Includes one suspected case of mesothelioma.</p> <p><sup>c</sup>According to the mortality study, which was restricted to deaths before January 1, 1978. The text of this study also noted 26 cases of mesothelioma diagnosed to January 1, 1979.</p> <p><sup>d</sup>Pleural mesotheliomas included in lung cancer total given by the authors but taken out of the lung cancer total for the purpose of this table.</p> <p><sup>e</sup>Authors stated that none of the cases were clearly attributable to asbestos exposure.</p> <p><sup>f</sup>Number of deaths 20 or more years from first exposure.</p> <p><sup>g</sup>Two cases not meeting criteria for entry into the cohort.</p> <p><sup>h</sup>Includes mesotheliomas.</p> <p><sup>i</sup>Peritoneal mesotheliomas included in gastrointestinal cancer total given by the authors but taken out of the gastrointestinal cancer total for the purpose of this table.</p> <p><sup>j</sup>"Best Evidence" figure.</p> <p><sup>m</sup>Minimal usage.</p> <p>NS means not stated.</p> <p>M means male.</p> <p>F means female.</p>												
<b>SOURCES:</b>	<table border="0"> <tr> <td data-bbox="262 834 453 859">J.C. McDonald, 1980:</td><td data-bbox="502 834 981 916">J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," <i>British Journal of Industrial Medicine</i> 37 (1980): 11-24.</td></tr> <tr> <td data-bbox="262 916 453 941">Nicholson, 1979:</td><td data-bbox="502 916 981 1040">William J. Nicholson et al., "Long-Term Mortality Experience of Chrysotile Miners and Millers in Thetford Mines, Quebec," <i>Annals of the New York Academy of Sciences</i> 330 (14 December 1979): 11-21.</td></tr> <tr> <td data-bbox="262 1040 453 1065">Rubino, 1979:</td><td data-bbox="502 1040 981 1148">G.F. Rubino et al., "Mortality of Chrysotile Asbestos Workers at the Balangero Mine, Northern Italy," <i>British Journal of Industrial Medicine</i> 36 (1979): 187-194.</td></tr> <tr> <td data-bbox="262 1148 453 1172">Hobbs, 1980:</td><td data-bbox="502 1148 981 1354">M.S.T. Hobbs et al., "The Incidence of Pneumoconiosis, Mesothelioma and Other Respiratory Cancer in Men Engaged in Mining and Milling Crocidolite in Western Australia," in <i>Biological Effects of Mineral Fibres</i>, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 615-625.</td></tr> <tr> <td data-bbox="262 1354 453 1379">Meurman, 1974:</td><td data-bbox="502 1354 981 1478">L.O. Meurman, R. Kiviluoto, and M. Hakama, "Mortality and Morbidity Among the Working Population of Anthophyllite Asbestos Miners in Finland," <i>British Journal of Industrial Medicine</i> 31:2 (April 1974): 105-112.</td></tr> <tr> <td data-bbox="262 1478 453 1503">Berry, 1983:</td><td data-bbox="502 1478 981 1538">Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using</td></tr> </table>	J.C. McDonald, 1980:	J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," <i>British Journal of Industrial Medicine</i> 37 (1980): 11-24.	Nicholson, 1979:	William J. Nicholson et al., "Long-Term Mortality Experience of Chrysotile Miners and Millers in Thetford Mines, Quebec," <i>Annals of the New York Academy of Sciences</i> 330 (14 December 1979): 11-21.	Rubino, 1979:	G.F. Rubino et al., "Mortality of Chrysotile Asbestos Workers at the Balangero Mine, Northern Italy," <i>British Journal of Industrial Medicine</i> 36 (1979): 187-194.	Hobbs, 1980:	M.S.T. Hobbs et al., "The Incidence of Pneumoconiosis, Mesothelioma and Other Respiratory Cancer in Men Engaged in Mining and Milling Crocidolite in Western Australia," in <i>Biological Effects of Mineral Fibres</i> , vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 615-625.	Meurman, 1974:	L.O. Meurman, R. Kiviluoto, and M. Hakama, "Mortality and Morbidity Among the Working Population of Anthophyllite Asbestos Miners in Finland," <i>British Journal of Industrial Medicine</i> 31:2 (April 1974): 105-112.	Berry, 1983:	Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using
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Berry, 1983:	Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using												

SOURCES:  
(continued)

- Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 1-7. See also, Muriel L. Newhouse, Geoffrey Berry, and Joseph W. Skidmore, "A Mortality Study of Workers Manufacturing Friction Materials with Chrysotile Asbestos," *Annals of Occupational Hygiene* 26:1-4 (1982): 899-909.
- A.D. McDonald, 1984: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant," *British Journal of Industrial Medicine*, in press, 1984.
- Peto, 1977: Julian Peto et al., "A Mortality Study Among Workers in an English Asbestos Factory," *British Journal of Industrial Medicine* 34 (1977): 169-173.
- Peto, 1980: Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 829-836.
- Dement, 1982: John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1-4 (1982): 869-887.
- A.D. McDonald, 1983: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," *British Journal of Industrial Medicine* 40 (1983): 361-367.
- A.D. McDonald, 1983: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," *British Journal of Industrial Medicine* 40 (1983): 368-374.
- Robinson, 1979: Cynthia F. Robinson, Richard A. Lemen, and Joseph K. Wagoner, "Mortality Patterns, 1940-1975 Among Workers Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility," in *Dusts and Disease*, eds. Richard A. Lemen and John M. Dement (Park Forest South, Illinois: Pathatox Publishers, Inc., 1979), pp. 131-143.
- Weill, 1979: Hans Weill, Janet Hughes, and Carmel Waggenpack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease* 120:2 (August 1979): 345-354.
- Finkelstein, 1983: Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983. (Mimeographed.)
- Thomas, 1982: H.F. Thomas et al., "Further Follow-up Study of Workers from an Asbestos Cement Factory," *British Journal of Industrial Medicine* 39:3 (August 1982): 273-276.

- SOURCES: Henderson and  
(continued) Enterline, 1979:
- Vivian L. Henderson and Philip E. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 117-126.
- Newhouse, 1979: Muriel L. Newhouse and Geoffrey Berry, "Patterns of Mortality in Asbestos Factory Workers in London," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 53-60.
- Jones, 1980: J.S.P. Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 637-653.
- Seidman, 1979: Herbert Seidman, Irving J. Selikoff, and E. Cuyler Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 61-89.
- Newhouse, 1979: Muriel L. Newhouse and Geoffrey Berry, "Patterns of Mortality in Asbestos Factory Workers in London," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 53-60.
- Selikoff, 1979: Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91-116.
- Selikoff, 1979: Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91-116.
- Rossiter, 1980: Charles E. Rossiter and Ruth M. Coles, "HM Dockyard, Devonport: 1947 Mortality Study," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 713-721.

Lancaster stands in stark contrast to the virtual absence of this disease at Charleston.

Thus, we conclude that while the health risks of asbestos are a function of industrial process, process alone does not provide a complete explanation of the incidence of disease that has been recorded. We turn, then, to a second factor that appears to have affected the incidence of asbestos-related disease: fibre type.

## C. The Effect of Fibre Type

### C.1 Introduction

The present Ontario Regulation Respecting Asbestos establishes control limits which clearly differentiate by fibre type.<sup>144</sup> In keeping with the recommendations of the U.K. Advisory Committee on Asbestos, the Ontario asbestos regulation reflects the view that of the three principal fibre types, chrysotile is the least hazardous and crocidolite the most hazardous, with amosite occupying an intermediate position. However, this view is one that has by no means gone unchallenged. There is perhaps no issue related to the health effects of asbestos that has evoked as much debate as the issue of whether the amphiboles, and particularly crocidolite, are more hazardous than chrysotile or whether they are all equally hazardous. At the time of our hearings, both the medical and the scientific communities, as well as regulatory agencies, appeared divided on this issue, and their division of opinion was quite evident during testimony before us. For example, in contrast to the position taken by the U.K. Advisory Committee,<sup>145</sup> Mr. Richard A. Lemen of NIOSH testified that the Institute as well as the United States regulatory agency, the Occupational Safety and Health Administration (OSHA), were of the opinion that the health evidence did not warrant any differentiation on the basis of fibre type.<sup>146</sup> Dr. William J. Nicholson and Mr. Julian Peto, among others, expressed similar views.<sup>147</sup> At the other end of the spectrum, Dr. J. Corbett McDonald and his associates were firmly of the view that amphibole exposure was considerably more hazardous than

<sup>144</sup> Regulation Respecting Asbestos, O. Reg. 570/82, ss. 4(1) and 4(2), made under the *Occupational Health and Safety Act*, R.S.O. 1980, c. 321.

<sup>145</sup> U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee*, paragraphs 176–211, pp. 74–78.

<sup>146</sup> RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, pp. 12–14.

<sup>147</sup> RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 78–79; and RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 61–63, 126–127. See also, RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, pp. 47–48; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 26 August 1981, Volume no. 30, pp. 54–55.



chrysotile exposure, and so much so that the amphiboles ought to be banned.<sup>148</sup>

We do not pretend that we have a definitive resolution to the scientific controversy over fibre type; our interest lies in whether the weight of the evidence supports differentiation for regulatory purposes. The evidence from various studies, both epidemiological and experimental, gives us a considerable amount of information about the relative hazards of chrysotile, crocidolite, and amosite. In order to sort out this evidence in a sensible manner, we believe it is helpful to consider the evidence for and against differentiation by fibre type in relation to each asbestos-related disease separately. The principal focus of our analysis is whether the amphiboles (crocidolite and amosite) are or are not more hazardous than chrysotile. We separately consider whether there is any evidentiary basis for differentiating between crocidolite and amosite. Finally, we consider briefly the evidence as to the health effects of anthophyllite, tremolite, and actinolite.

## C.2 The Evidence in Relation to Asbestosis

The epidemiological evidence that might indicate a differentiation according to fibre type in relation to the development of asbestosis is in our judgement minimal. There does exist one study providing an internal comparison which suggests that crocidolite may have a greater fibrogenic effect than chrysotile. This is Dr. Weill's study,<sup>149</sup> the results of which were cited in the report of the U.K. Advisory Committee.<sup>150</sup> Dr. Weill examined the clinical, radiographic, and physiological indicators of lung disease among two groups of workers who had spent 20 to 30 years in the asbestos-cement industry in New Orleans. One group had exposure to chrysotile, silica, and crocidolite in the pipe-making area. The other group had no crocidolite exposure at all, having worked only with chrysotile. Dr. Weill found that the crocidolite-exposed group had a significantly increased prevalence of small irregular opacities and pleural thickening, smaller lung volumes, lower

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<sup>148</sup>RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 46-47; and 25 June 1981, Volume no. 13, p. 53.

<sup>149</sup>Hans Weill et al., "Lung Function Consequences of Dust Exposure in Asbestos Cement Manufacturing Plants," *Archives of Environmental Health* 30:2 (February 1975): 88-97. See also, Hans Weill et al., "Differences in Lung Effects Resulting from Chrysotile and Crocidolite Exposure," in *Inhaled Particles IV*, Part 2, ed. W.H. Walton (Oxford: Pergamon Press, 1977), pp. 789-798.

<sup>150</sup>Acheson and Gardner, "The Ill Effects of Asbestos on Health," paragraph 121, p. 26.

forced expiratory flow, reduced pulmonary diffusion, and a greater prevalence of clubbing than the group not exposed to crocidolite.<sup>151</sup> The radiographic characteristics of the two groups of workers are set out in Table 5.19.

On the other hand, a recent report by Dr. Margaret R. Becklake and her colleagues of asbestos workers in two plants in Montreal — a textile plant using only chrysotile and a plant manufacturing insulation and cement products in which some amosite and crocidolite had been used along with chrysotile — failed to demonstrate that fibre type had a significant influence on the prevalence of respiratory symptoms and functional abnormalities.<sup>152</sup> Rather, the findings suggested that process, not fibre type, was the dominant factor affecting the results.<sup>153</sup> (See Table 5.20.) Similarly, in parallel investigations by Dr. A.D. McDonald et al. of United States asbestos factories, exposure-response relationships for asbestosis in the two textile plants studied — the one in Charleston and the other in Pennsylvania — were similar, notwithstanding that the former used only chrysotile while the latter used both crocidolite and amosite in addition to chrysotile.<sup>154</sup>

The epidemiological findings of Dr. Becklake and Dr. A.D. McDonald are supported by animal inhalation experiments. These experiments have not only demonstrated that all fibre types are fibrogenic, but have suggested that if anything chrysotile was not less but more fibrogenic than the amphiboles. For example, in 1974, Dr. J. Christopher Wagner and his associates reported the results of two experiments in which rats were exposed by inhalation to dust clouds of the UICC (Union Internationale Contre le Cancer) standard reference samples.<sup>155</sup> All samples produced

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<sup>151</sup> Weill et al., "Differences in Lung Effects Resulting from Chrysotile and Crocidolite Exposure," p. 789. In his evidence, Dr. Weill suggested that the greater fibrogenic effect attributable to crocidolite could have been due to the physical dimension of the fibre or could simply have been due to the dustiness of the fibre. See RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 36–37.

<sup>152</sup> Margaret R. Becklake et al., "Exposure to Asbestos and Respiratory Abnormality: The Influence of Fibre Type and Nature of Exposure," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 763–768.

<sup>153</sup> *Ibid.* The authors carried out health surveys on workers in two plants in the Montreal region: a textile plant in which only chrysotile had been used and a plant manufacturing insulation and cement products in which chrysotile was the dominant fibre used, but in which both amosite and crocidolite had also been used. The results were compared with results previously obtained in the chrysotile mining industry in Quebec. Despite apparently shorter exposure to lower dust levels, asbestos exposure in the manufacturing sector had more effect on respiratory status than exposure in mining and milling. Between-plant differences were less than between-process differences.

<sup>154</sup> A.D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," p. 373.

<sup>155</sup> J. Christopher Wagner et al., "The Effects of the Inhalation of Asbestos in Rats," *British Journal of Cancer* 29:3 (March 1974): 252–269.

**Table 5.19**  
**Radiographic Characteristics**

<b>Radiographic Abnormality</b>	<b>75% or More in Pipe Area (108)</b>	<b>Never Worked in Pipe Area (100)</b>	
Small rounded opacities 1/1+	6 (5.6%)	4 (4%)	Non-significant
Small irregular opacities 1/1+	11 (10.2%)	2 (2%)	P = 0.02
Pleural thickening 1+	34 (31%)	15 (15%)	P = 0.006

SOURCE: Hans Weill et al., "Differences in Lung Effects Resulting from Chrysotile and Crocidolite Exposure," in *Inhaled Particles IV*, Part 2, ed. W.H. Walton (Oxford: Pergamon Press, 1977), Table 2, p. 792.

Table 5.20

**Prevalence (%) of Abnormality, Adjusted for Age and Smoking Status,  
in Three Workforces Exposed to Asbestos**

<b>Radiographic Abnormality</b>	<b>Insulation and Cement Products</b>	<b>Textiles</b>	<b>Mining and Milling</b>
Rounded opacities	1.5	1.6	1.8
Irregular opacities	19.7	20.9	6.6
Pleural thickening	20.8	15.7	4.7
Pleural calcification	3.4	2.5	3.4

SOURCE: Presented during Ontario, Royal Commission on Asbestos, Transcript of Public Hearings, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20. Revised during telephone communication between Dr. Margaret R. Becklake, Department of Epidemiology and Health, McGill University, Montreal, Quebec and Royal Commission on Asbestos Staff, 28 October 1983.



asbestosis which continued to progress after removal from exposure. There was no evidence of less asbestosis among the rats exposed to chrysotile than those exposed to the amphiboles even though much less dust was retained in the lungs of the chrysotile-exposed animals. (See Table 5.21.) In a more recent inhalation study by Dr. John M.G. Davis, the chrysotile dust clouds produced far more interstitial pulmonary fibrosis than the amphibole clouds when the animals were examined 29 months after the start of inhalation.<sup>156</sup> (See Table 5.22.) These results appear to us representative of the animal inhalation studies that have investigated the issue of fibre type in relation to asbestosis.<sup>157</sup>

### C.3 The Evidence in Relation to Lung Cancer

When we turn to the incidence of lung cancer, there is more evidence than there is with respect to asbestosis. However, as we shall see, this evidence does not appear to us to yield any consistent pattern that, absent differences in industrial process or other factors, would implicate the amphiboles per se as more hazardous.

Perhaps the strongest evidence against the amphiboles comes from the experience of the factory and maintenance workers studied by Dr. Weill and by Dr. Enterline in their respective cohort studies. In order to assess the potential influence of fibre type on the risk of lung cancer, Dr. Weill, as we have already observed, compared the mortality experience of that part of his cohort exposed to chrysotile only (4,201 workers) with two groups of workers exposed to crocidolite as well: those with steady exposure in the pipe plant (1,004 workers) and those with intermittent exposure to crocidolite through occasional maintenance work in the area (235 workers). Follow-up and exposure data for these groups were roughly similar.<sup>158</sup> Dr.

<sup>156</sup> John M.G. Davis, "The Use of Animal Inhalation Experiments in the Study of Asbestos Bioeffects," *Staub-Reinhalt. Luft* 40:10 (October 1980): 453-456.

<sup>157</sup> The animal evidence is reviewed in detail in U.S., National Institute for Occupational Safety and Health, *Revised Recommended Asbestos Standard*, prepared by Richard A. Lemen and John M. Dement (Washington, D.C.: U.S. Department of Health, Education and Welfare, December 1976); World Health Organization, International Agency for Research on Cancer Working Group, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, Lyon, France: 14-17 December 1976 (Lyon: IARC, 1977); and John M.G. Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature* (Edinburgh: Institute of Occupational Medicine, December 1980). Dr. Davis concluded from his review of the literature that "Animal experimental studies suggest that chrysotile is more fibrogenic and produces more bronchial carcinomas following inhalation than the amphibole varieties." (p. 17.) See also, John M.G. Davis, "The Use of Animal Experiments in the Study of Asbestos Bioeffects," *Hefte Unfallheilkd* 126 (November 1975): 564-574.

<sup>158</sup> Weill, Hughes, and Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," pp. 351-352.

Table 5.21  
Mean Asbestosis Scores\* of Sacrificed Rats

Length of Exposure	Amosite	Anthophyllite	Crocidolite	Chrysotile (Canadian)	Chrysotile (Rhodesian)	Control
8 weeks	2.0	2.0	2.0	2.0	2.7	1.3
3 months	2.5	2.7	2.8	2.7	3.0	1.3
6 months	2.2	3.2	2.6	3.0	2.6	1.2
12 months	4.0	5.2	4.3	4.3	4.3	—
18 months	—	—	—	—	—	1.2
24 months	4.3	6.2	4.8	6.0	5.8	1.8
6 months (after 18 months non-exposure)	3.2	5.0	3.7	5.5	3.7	—

Notes: \*1: nil; 2: minimal; 4: slight; 6: moderate; 8: severe.

SOURCE: J. Christopher Wagner et al., "The Effects of the Inhalation of Asbestos in Rats," *British Journal of Cancer* 29:3 (March 1974): 262 (Table V).

Table 5.22

The Effects of Fibre Number on the Levels of Lung Damage Produced  
by the UICC Samples of Chrysotile, Amosite, and Crocidolite

Asbestos Type	Dust Cloud Mass (mg/m <sup>3</sup> )	Fibre Number (ml <sup>-1</sup> )	Interstitial Fibrosis at 29 Months (%)	Lung Tumours	
				Benign	Malignant
Chrysotile	10	1,950	9.1	7	8
Crocidolite	10	860	1.4	1	0
Amosite	10	550	2.6	2	0
Chrysotile	2	390	3.8	6	2
Crocidolite	5	430	0.8	2	0

SOURCE: John M. G. Davis, "The Use of Animal Inhalation Experiments in the Study of Asbestos Bioeffects," *Staub-Reinhalt. Luft* 40:10 (October 1980): 454 (Table 1).

Weill reported that the pattern which emerged from comparing SMRs indicated that the crocidolite-exposed groups, and particularly the maintenance workers, had a higher risk of lung cancer than the solely chrysotile-exposed group. The results of Dr. Weill's comparison are summarized in Table 5.23. Even accepting the possible confounding effect of intermittent, high exposures for the maintenance workers, the influence of crocidolite appears to have been significant.<sup>159</sup> Similarly, Henderson and Enterline, in their factory retirees study, were able to compare the differences in lung cancer mortality between those in their cohort working in the asbestos-cement industry who were exposed both to crocidolite and chrysotile while manufacturing asbestos-cement pipe and those exposed only to chrysotile while manufacturing asbestos-cement shingles and sheets.<sup>160</sup> The authors reported that the men employed in these two segments of the industry had much in common: exposure to silica occurred in both cases; the levels of dust exposure were similar and occurred during the same processes; and often the two types of products were produced at a single location. In short, the main difference in the manufacture of these two types of products appeared to be the fibre types used. To repeat the data from Henderson and Enterline's most recent report, the 97 crocidolite-exposed asbestos-cement pipe workers had a mean exposure of 230 mppcf-yrs and a lung cancer SMR of 521.7; the 305 asbestos-cement shingle and sheet workers exposed only to chrysotile had a mean exposure of 255 mppcf-yrs and a lung cancer SMR of only 230.8.<sup>161</sup> With regard to amosite asbestos, the results were far less conclusive. For the 58 retirees from the single plant where only amosite was used, the SMR for lung cancer was high at 363.6, but so was the mean exposure of 330 mppcf-yrs; moreover, in those plants using amosite in combination with other types of asbestos, the risk of lung cancer was not much different than that found among the retirees exposed to chrysotile alone. (See Table 5.24.)

Some further indication of a different health hazard, at least as between chrysotile and crocidolite in terms of lung cancer mortality, comes from a comparison of the experience of the Quebec chrysotile miners and the Western Australian crocidolite miners. For example, the lung cancer SMR, 20 or more years from first employment in Quebec for all those employees who had worked one year or more, was 128;<sup>162</sup> the lung cancer SMR, 15 or more years from first employment in Western Australia for all

<sup>159</sup>RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 37-38.

<sup>160</sup>Henderson and Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," pp. 122-124.

<sup>161</sup>*Ibid.*, p. 121. It may also be observed that mortality rates for pneumoconiosis-pulmonary fibrosis (generally asbestosis) did not follow a similar pattern. Those workers exposed to chrysotile alone had a mortality rate per 100,000 of 396.3, whereas those workers exposed both to chrysotile and crocidolite had a corresponding mortality rate of 215.0.

<sup>162</sup>J. Corbett McDonald, "Asbestos-Related Disease: An Epidemiological Review," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 588-589.



**Table 5.23**  
**Standard Mortality Ratios for Respiratory Malignancy by Fibre Type**

Type of Exposure	Total Fibre Exposure Within 20 Years of Initial Employment (mppcf-mos)*			
	≤20	20-200	>200	Total
No crocidolite exposure	12/21.4 56	10/13.0 77	8/4.4 182	30/38.8 77
Intermittent exposure to crocidolite in pipe plant	2/0.2 1,000**	0/0.7 0	5/1.4 357**	7/2.3 304**
Steady employment in pipe plant with crocidolite exposure	1/1.0 100	1/1.9 53	7/2.9 241**	9/5.8 155

**Notes:** \*Million particles per cubic foot-months.

\*\*Significant at  $p < 0.05$ .

SOURCE: Hans Weill, Janet Hughes, and Carmel Waggenpack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease* 120:2 (August 1979): 352 (Table 8).

Table 5.24  
Respiratory Cancer Mortality by Type of Asbestos,  
1,075 Men Retiring 1941-1967 and Followed Through 1973

Type of Asbestos	Number of Men	Mean Exposure (mppcf-yrs)	Predicted SMR	Observed		95% Confidence Limits for	
				Deaths	SMR	Observed	SMR
Amosite only	58	330	317.1	4	363.6	99.1-	929.9
Chrysotile only	754	244	260.6	40	246.9	176.4-	336.4
Amosite and chrysotile	98	274	280.3	4	181.8	49.5-	465.0
Chrysotile and crocidolite	112	209	237.5	12	461.5	237.9-	805.4
Asbestos-cement pipe	83	219	244.1	11	578.9	289.4-	1,035.6
Others	29	180	218.4	1	142.9	3.6-	793.9
Amosite, chrysotile, and crocidolite	53	277	282.3	3	250.0	51.5-	731.0
Asbestos-cement pipe	14	296	294.8	1	250.0	6.3-	1,388.9
Others	39	270	277.7	2	250.0	30.3-	902.5

SOURCE: Vivian L. Henderson and Philip E. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 122 (Table 4).

employees who had similarly worked one year or more, was nearly double at 246.<sup>163</sup> Such a comparison between different cohorts with different dust exposures is admittedly fraught with uncertainty, but it does provide some evidence of the relative health risks between fibre types at least in asbestos mining.

And yet, a similar comparison between textile operations suggests quite a different conclusion. As we have already observed, of the three United States asbestos plants studied by Dr. A.D. McDonald, two manufactured textiles: the Charleston plant (studied by Dr. Dement as well), using only chrysotile, and the Pennsylvania plant, using all three main fibre types. In both plants, the cohorts were constituted on the same basis; in both plants, the employees on average worked for similar lengths of time; their smoking habits did not appear to differ greatly, and if anything the average exposures in Pennsylvania were higher at 2.32 mppcf as compared to 1.80 mppcf in Charleston. Yet the SMR for lung cancer 20 or more years from first employment for Dr. A.D. McDonald's entire cohort in Charleston was 199.5, while in Pennsylvania it was only 105; and in the highest dust exposure category, the figure was 1,031.9 in Charleston and 416.1 in Pennsylvania.<sup>164</sup> Not only was the lung cancer risk higher in the plant using chrysotile only, it is fair to say that amongst all of the various cohort studies that have been reported, none has had a higher lung cancer mortality rate than the Charleston chrysotile textile plant. This in itself is strong evidence of the fact that there is no consistent association between fibre type and health effects insofar as lung cancer mortality is concerned.

Moreover, as with asbestosis, the results of the animal evidence in relation to lung cancer do not indicate that chrysotile is any less hazardous than the amphiboles. Indeed, in several inhalation studies, chrysotile has had a rather worse malignant lung tumour record than the amphiboles. Thus, in the large inhalation experiment published by Wagner et al. in 1974 in which rats had been treated with UICC standard reference samples, Rhodesian chrysotile produced by far the greatest number of lung tumours, followed in order by Canadian chrysotile, crocidolite, and amosite.<sup>165</sup> The results of this study as they relate to the production of lung tumours are summarized in Table 5.25. In the later inhalation experiment by Dr. Davis,

<sup>163</sup> Hobbs et al., "The Incidence of Pneumoconiosis, Mesothelioma and Other Respiratory Cancer in Men Engaged in Mining and Milling Crocidolite in Western Australia," p. 622.

<sup>164</sup> A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," Tables 4 and 5, p. 364; and A.D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," Tables 4 and 5, p. 371.

<sup>165</sup> Wagner et al., "The Effects of the Inhalation of Asbestos in Rats."

**Table 5.25**  
**Number of Animals with Lung Tumours**  
**or Mesotheliomata**

<b>Exposure</b>	<b>Number of Rats at Risk*</b>	<b>Number of Malignant Lung Tumours</b>	<b>Number of Mesotheliomas</b>
Amosite	146	11	1
Anthophyllite	145	16	2
Crocidolite	141	16	4
Chrysotile (Canadian)	137	17	4
Chrysotile (Rhodesian)	144	30	0
Control	126	0	0

**Note:** \* Rats which survived at least 300 days after start of exposure.

**SOURCE:** Adapted from: J. Christopher Wagner et al., "The Effects of the Inhalation of Asbestos in Rats," *British Journal of Cancer* 29:3 (March 1974): 264 (Table VII).



all of the malignant lung tumours developed in the chrysotile-treated animals; none were produced by either crocidolite or amosite dust clouds.<sup>166</sup> (See Table 5.22 in this chapter.)

## C.4 The Evidence in Relation to Mesothelioma

However, it is neither the incidence of asbestosis nor of lung cancer which has prompted the concern about amphibole asbestos exposure. Rather, the principal indictment against crocidolite and amosite has been based upon the demonstrated incidence of mesothelioma among cohorts of asbestos workers exposed to these fibre types and the comparatively rare occurrence of the tumour in chrysotile-exposed populations.

Perhaps the most compelling evidence comes from the experience of a small group of gas mask workers employed during World War II in both Canada and England. The health experience of the Canadian workers has been studied by Dr. A.D. McDonald and Dr. J.C. McDonald, who reported on a cohort of 199 persons exposed to crocidolite between 1939 and 1942 in three separate factories.<sup>167</sup> In two of the plants, both located in Montreal, filter pads containing pure crocidolite were made, and in a third plant, located in Ottawa, these filters were packed into cannisters which were assembled into gas masks for use by the Canadian army. These cannisters contained crocidolite, 20% by weight, and wool, 80% by weight. Of the 199 employees, 174 were traced to the end of 1975, by which time 56 had died. Out of these 56 deaths, 9 were from mesothelioma (and, it may be noted, 7 from lung cancer). This statistic provides a stark contrast to the experience of the Quebec chrysotile miners. In the Quebec mines, a cohort of 11,379 workers, many of whom were long-term employees, yielded but 11 deaths from mesothelioma. Among the gas mask workers, a cohort approximately 1/60 in size, none of whom were employed longer than 3 years, produced almost the same number of mesotheliomas (and, it may be observed parenthetically, a lung cancer risk twice as high). There was also an important difference in the site of the tumours between the two cohorts: all 11 of the mesotheliomas among the chrysotile miners were pleural, whereas 6 of the 9 mesotheliomas among the crocidolite gas mask workers were peritoneal.<sup>168</sup> Table 5.26 compares malignant disease in the chrysotile miners and millers and in the crocidolite gas mask workers to the end of 1975.

<sup>166</sup> Davis, "The Use of Animal Experiments in the Study of Asbestos Bioeffects," p. 454.

<sup>167</sup> Alison D. McDonald and J. Corbett McDonald, "Mesothelioma After Crocidolite Exposure During Gas Mask Manufacture," *Environmental Research* 17 (December 1978): 340-346.

<sup>168</sup> Ibid., pp. 343-344.

**Table 5.26**  
**Malignant Disease in Chrysotile Miners**  
**and Millers and in Crocidolite Gas Mask Filter**  
**Workers to the End of 1975**

	<b>Crocidolite Gas Mask Filter Workers</b>	<b>Chrysotile Miners and Millers</b>
Total cohort	199 (55% male)	11,379 (96% male)
Traced	174	10,259
Dead	56	4,247 (since 1935)
Cancer of the lung or bronchus	7 (13%)	242 (6%)
Mesothelioma	9 (16%)*, ***	11 (0.26%)*, ***
Other malignant disease	7 (13%)	632 (14%)

**Notes:** \*Four certified; two diagnosed at autopsy; three after pathologist's review.

\*\*Eight certified, one of which was not thought by the pathologist to be a mesothelioma; three diagnosed at autopsy.

\*\*\* Two cases were common to both series \* and \*\* above.

SOURCE: Alison D. McDonald and J. Corbett McDonald, "Mesothelioma After Crocidolite Exposure During Gas Mask Manufacture," *Environmental Research* 17 (December 1978): 344 (Table 3).

The disease record of the Canadian gas mask workers was approximately matched by a group of female employees assembling gas masks at a factory in Nottingham, England, during the war.<sup>169</sup> In fact, there were two types of gas masks made at the English factory: military masks, which as in Canada used crocidolite filters and were assembled over a period of 4½ years; and civilian masks, which used chrysotile filters and were made on a limited scale for a period of 5 months. The mortality experience of these workers has been studied and reported on by Jones, Pooley, and Smith beginning in 1976.<sup>170</sup> Due to the small number of workers producing civilian masks and their limited asbestos exposure, the authors declined to draw any conclusion about their health experience. But the crocidolite-exposed women responsible for assembling military masks have been followed to the end of 1978. Of the cohort of 951 employees, only 578 have been traced. Among this group, there have been 166 deaths, of which 17 or fully 10% have died of mesothelioma. The authors reported that 13 of the mesotheliomas were pleural and only 4 peritoneal, a somewhat different proportion than found in the Canadian cohort.<sup>171</sup>

The incidence of mesothelioma among the crocidolite-exposed gas mask workers, compelling as it may be, does not stand alone. The Western Australian crocidolite miners have produced more than twice as many mesotheliomas as the Quebec chrysotile miners in a cohort little more than half the size.<sup>172</sup> The incidence of mesothelioma at and in the vicinity of the Cape crocidolite mines in South Africa, while not the subject of any systematic study, is on its face dramatically different from the experience of the asbestos miners and residents of the Eastern Townships in Quebec. In addition to the substantial number of mesotheliomas occurring among the Cape crocidolite miners themselves, Professor Webster, in the period 1956–1982, reported 178 cases of mesothelioma due to domestic or

<sup>169</sup> J.S.P. Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 637–653.

<sup>170</sup> J.S.P. Jones, F.D. Pooley, and P.G. Smith, "Factory Populations Exposed to Crocidolite Asbestos — A Continuing Survey," in *Environmental Pollution and Carcinogenic Risks*, eds. C. Rosenfeld and W. Davis, IARC Scientific Publications, no. 13 (Lyon, France: International Agency for Research on Cancer, 1976), pp. 117–120.

<sup>171</sup> Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," p. 645. Among these female workers, there was also an excess of lung cancer, with 12 observed as against 6.3 expected, but no excess of any other type of malignant disease, including gastrointestinal cancer.

<sup>172</sup> See Section B.1 of this chapter.

neighbourhood exposure.<sup>173</sup> By contrast, despite the extensive mining of chrysotile asbestos in Quebec and the extensive investigation of the incidence of mesothelioma in that province, there is a notably lower incidence of the disease among those living or working in the mining area. In her study of malignant mesothelioma in Quebec between 1960 and 1978, Dr. A.D. McDonald reported 6 cases among family members of Quebec miners and millers and 2 persons with the disease who had lived within 20 miles of the chrysotile mines.<sup>174</sup>

This evidence is admittedly circumstantial. But there is more direct evidence. At least 8 of the 10 mesotheliomas (all of which were pleural) at the Ferodo plant have been attributed by Berry and Newhouse to the crocidolite exposure in a plant using predominantly chrysotile and where otherwise there was little or no evidence of disease.<sup>175</sup> In A.D. McDonald's study of the three United States asbestos factories, among the over 2,000 deaths in the two factories using chrysotile, there has been only one mesothelioma, whereas among the 1,400 deaths in the factory using a combination of chrysotile and the amphiboles, there have been no less than 14 mesotheliomas (10 pleural and 4 peritoneal), with a distinct possibility of other unrecognized peritoneal tumours.<sup>176</sup> The amphibole-exposed naval shipyard workers in England studied by Rossiter and Coles produced 31 mesotheliomas (and 9 deaths from asbestosis) but otherwise little evidence of increased mortality among the 1,043 deaths in the total cohort, suggesting that while the exposure of these workers may not have been heavy, it

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<sup>173</sup> Letter from Professor Ian Webster to the Royal Commission on Asbestos, 14 July 1983. See also, J.M. Talent et al., "A Survey of Black Mineworkers of the Cape Crocidolite Mines," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 723-729. It would appear that over 90% of the environmental cases reported by Professor Webster have been exposed to Cape crocidolite. Telephone communication between Professor Ian Webster and Royal Commission on Asbestos Staff, 27 October 1983. See also, J. Corbett McDonald, "Exposure Relationships and Malignant Mesothelioma," in *Proceedings of Asbestos Symposium*, Johannesburg, South Africa: 3-7 October 1977, ed. H.W. Glen (Randburg: National Institute for Metallurgy, 1978): 79. (Discussion.)

<sup>174</sup> A.D. McDonald, "Malignant Mesothelioma in Quebec," pp. 675-676. In the article, Dr. A.D. McDonald also compared the incidence of mesothelioma among the chrysotile miners and millers with the incidence of mesothelioma in four asbestos factories in the province of Quebec. Twenty-two cases of mesothelioma occurred within 50 years of employment in three of the factories, all of which used amphibole asbestos at various points in time and each of which apparently had an annual workforce of under 400. There were no cases of mesothelioma in the fourth factory (with an approximate annual employment of 140) which used only chrysotile and, at the time of the article, 16 cases of mesothelioma attributable to chrysotile exposure in the Quebec mines and mills, whose average annual employment was approximately 6,000.

<sup>175</sup> Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos."

<sup>176</sup> See Sections B.2 and B.3 of this chapter.



was particularly likely to induce mesothelioma.<sup>177</sup> (See Table 5.27.) There were 21 mesotheliomas at the Scarborough asbestos-cement factory studied by Finkelstein (of which 9 were peritoneal), all but one of which had crocidolite exposure; whereas at the Cardiff asbestos-cement factory investigated by Thomas et al. at which only chrysotile was used, save for a two-year period in the 1930s when there was some crocidolite, there were only 2 mesotheliomas (both pleural) in a cohort approximately 3 times as large as that at Scarborough, and both of those likely had crocidolite exposure.<sup>178</sup>

Among the cohort of 820 amosite insulation workers at Paterson, New Jersey, there have been (according to best evidence) 14 mesotheliomas, half of them peritoneal.<sup>179</sup> And, finally, of 2,271 deaths among the North American insulators exposed to chrysotile and amosite and studied by Dr. Selikoff, 175 died of mesothelioma (112 peritoneal and 63 pleural).<sup>180</sup> Again, to put this last statistic in perspective, the cohort of insulators studied by Dr. Selikoff had approximately one-half the number of deaths as occurred among the Quebec miners, but 16 times as many mesotheliomas.

The epidemiological evidence, at first blush overwhelming, should at least be qualified to some extent. In most instances, there is little or no direct evidence of the fibre concentrations to which the various cohorts we have discussed have been exposed. Accordingly, there is at least the possibility that what may appear to be the effect of fibre type may in whole or in part be the effect of high exposure levels. What appears to be a low incidence of mesothelioma in a particular cohort may reflect not the fibre type that was used, but the fact that exposure levels in the cohort were such

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<sup>177</sup>Charles E. Rossiter and Ruth M. Coles, "HM Dockyard, Devonport: 1947 Mortality Study," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 713–721. See also, J.C. McDonald, "Asbestos-Related Disease: An Epidemiological Review," p. 592; P.G. Harries, "Experience with Asbestos Disease and Its Control in Great Britain's Naval Dockyards," *Environmental Research* 11:2 (April 1976): 261–267; and Geoffrey Sheers and Ruth M. Coles, "Mesothelioma Risks in a Naval Dockyard," *Archives of Environmental Health* 35:5 (September/October 1980): 276–282. Crocidolite asbestos was widely applied by a spray process for environmental insulation and fire protection between approximately 1940 and the early 1960s. Between 1950 and 1963, there was a large increase in the amount of amosite used for machinery insulation. Harries noted that there had been 55 deaths from mesothelioma between 1964 and 1973 at the Royal Navy Dockyard at Plymouth, of which all but one were pleural. Sheers and Coles reported 108 cases of mesothelioma at this dockyard between 1964 and mid-1978, of which all but 3 were pleural.

<sup>178</sup>See Section B.4 of this chapter.

<sup>179</sup>See Section B.5 of this chapter. Even so, the ratio of mesothelioma cases to lung cancer cases is quite low in this cohort. Dr. A.D. McDonald in her testimony suggested this was likely because the Paterson, New Jersey, workers were, as a group, much older than is usual. RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 59–60.

<sup>180</sup>See Section B.6 of this chapter.

**Table 5.27**  
**Observed and Expected Deaths, by Cause, Among Dockyard Workers**

Cause of Death	Number of Deaths	England and Wales		South-west Estimates**	
		Expected	SMR*	Expected	SMR
All deaths	1,043	1,081.2	96	998.4	104
All cancers	265	282.1	94	255.6	104
Mesothelioma	31***	0.5	6,400	0.4	7,700 P<<0.001
Lung cancer	84***	119.7	70	100.3	84
Gastrointestinal cancer	63	83.3	76	76.2	83
Others	87†	78.6	111	78.7	111
Asbestosis, pulmonary fibrosis	9	0.03	27,000	0.03	32,000 P<<0.001
Other respiratory diseases	67	97.3	69	81.9	82
Circulatory diseases	518†	464.2	112	440.8	118 P<0.001
Other causes	174	237.6	73	220.1	79 P<0.01
Unknown causes	10	—	—	—	—

**Notes:** \* SMR means standardized mortality ratio.

\*\* Estimated death rates in south-west Britain (including Devonport).

\*\*\* Three also had asbestosis.

† One also had asbestosis.

SOURCE: Charles E. Rossiter and Ruth M. Coles, "HM Dockyard, Devonport: 1947 Mortality Study," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), Table 2, p. 717.

that, overall, there was not much disease. There were clearly few mesotheliomas among the Quebec miners given the size of the cohort, but the overall excess of lung cancer was also relatively low. Mr. Peto has in fact demonstrated that the ratio of excess lung cancer to mesothelioma (at least to pleural mesothelioma) is quite similar among most studies irrespective of what fibre type or types have been utilized.<sup>181</sup> (See Table 5.28.) Still, this ratio does not hold either for the English shipyard workers studied by Rossiter and Coles or for the Charleston textile workers.<sup>182</sup>

Further, among factory workers exposed to mixed fibre types, it may be inappropriate to attribute the incidence of mesothelioma to amphibole exposure when in fact chrysotile is invariably the predominant fibre used.<sup>183</sup> Mr. Peto has argued that the crocidolite exposure at Rochdale was minimal, yet the incidence of pleural mesothelioma was similar to that of the East London asbestos plant studied by Newhouse and Berry where crocidolite exposure was heavy, thus suggesting it is unlikely that crocidolite alone was responsible for the mesothelial tumours at Rochdale.<sup>184</sup> Further, the lack of

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<sup>181</sup> RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 61–63. See also, J. Corbett McDonald and Alison D. McDonald, “Mesothelioma as an Index of Asbestos Impact,” in *Banbury Report 9: Quantification of Occupational Cancer*, eds. Richard Peto and Marvin Schneiderman ([Cold Spring Harbor, New York]: Cold Spring Harbor Laboratory, 1981), Table 2, pp. 76–77.

<sup>182</sup> In the Rossiter and Coles study, there were 31 cases of mesothelioma (likely all pleural) and no excess lung cancer. Dr. Dement had no cases of pleural mesothelioma, one peritoneal mesothelioma, and 18.5 excess lung cancer cases. Mr. Peto himself indicated in his testimony that the Rossiter and Coles study was difficult to explain. RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 61–63.

<sup>183</sup> Julian Peto, Herbert Seidman, and Irving J. Selikoff, “Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment,” *British Journal of Cancer* 45 (1982): 124–135. The authors indicated that amphiboles are largely responsible for asbestos-related peritoneal mesothelioma, but went on to state:

It may therefore be dangerously optimistic to attribute the substantial incidence of pleural mesothelioma among chrysotile factory workers to occasional crocidolite exposure, merely because mesothelioma is rare among chrysotile miners. . . . The overall excess of lung cancer is also relatively low among chrysotile miners, and the only safe conclusion must be that dose-response relationships cannot be expected to apply outside the environment in which they are established, at least until the range of fibre sizes to be included in the fibre count has been chosen less arbitrarily. (pp. 134–135.)

<sup>184</sup> Peto, “The Hygiene Standard for Chrysotile Asbestos,” p. 488. See also, Muriel L. Newhouse and Geoffrey Berry, “Patterns of Mortality in Asbestos Factory Workers in London,” *Annals of the New York Academy of Sciences* 330 (14 December 1979): 53–60. This East London asbestos factory, studied by Newhouse and Berry, opened in 1913, at first producing chiefly asbestos textiles but later producing insulation materials and a variety of other products. Crocidolite asbestos was used until the late 1950s and chrysotile and amosite were also used. The factory closed in 1968. In the latest mortality study, the authors reported 775 deaths among the male workers, a cohort of 4,600. An analysis of the 545 deaths that occurred among the factory workers, excluding laggings, revealed 103 deaths from lung cancer, an excess of 60 over expected; and 46 deaths from mesothelioma, of which 19 were pleural.

Table 5.28  
Ratio of Excess Lung Cancer to Mesothelioma

Fibre	Lung and Pleural Cancer (A)			Mesothelioma (B)	Mesothelioma/Excess (B)/(A)
	O	E	O-E	O	
Chrysotile and Crocidolite (Enterline)	14 49	5.2 18.1	8.8 30.9	3 2	34% 6%
Insulation (Selikoff)	533	105.6	427.4	175	41%
Crocidolite Mine (Hobbs)	56	28.7	27.3	9	33%
Chrysotile and Crocidolite(?) (Peto)	47	23.3	23.7	7	30%
Chrysotile Mine (J.C. McDonald)	230	184.0	46.0	11	24%
Mixed (Newhouse)	180	48.8	131.2	56	43%
Amosite (Seidman)	116	19.1	96.9	16	17%

SOURCE: Ontario, Royal Commission on Asbestos, Exhibit II-37, Tab 12, in RCA Transcript of Public Hearings, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 10, 61-63: Julian Peto, Assorted Transparencies. (Mimeographed.)



reported mesotheliomas among the Transvaal amosite and crocidolite miners provides some evidence that the amphiboles do not always produce a high incidence of this tumour.

And yet, even with these qualifications, there are three inescapable conclusions that in our judgement emerge from the epidemiological evidence. First, while chrysotile is capable of causing mesothelioma in humans, the incidence of mesothelioma among chrysotile-exposed cohorts has been relatively low. No better example may be found than the Charleston textile plant with an extraordinarily high incidence of lung cancer and yet but one mesothelioma. Second, amphibole exposure is at least capable of causing a significant incidence of mesothelioma in humans. The experiences of the gas mask workers, the North American insulators, and the English shipyard workers by themselves are eloquent testimony to this proposition. Third, the amphiboles appear to be almost solely responsible for the incidence of peritoneal (as opposed to pleural) mesothelioma among asbestos workers.<sup>185</sup> Almost without exception, the mesotheliomas that have been diagnosed in cohorts exposed only to chrysotile have been pleural.<sup>186</sup> Exposure to the amphiboles has not produced any consistent pattern. On the one hand, the mesotheliomas in the Western Australian crocidolite mines and at the English dockyards were nearly all pleural. On the other hand, two-thirds of the tumours among the Canadian gas mask workers and the North American insulators were peritoneal.<sup>187</sup> On balance, however, the observed incidence of tumours of the peritoneum has been associated with amphibole exposure. The possibility remains that this site is particularly susceptible to amosite exposure as evidenced by the experience of the amosite factory workers at Paterson, New Jersey, and the North American insulators.

The epidemiological evidence from cohort studies and case-series reports of the effect of fibre type on the incidence of mesothelioma is supported by the lung tissue studies that have been reported in the literature. For example, Jones et al. reported on a study of 93 cases of persons dying from mesothelioma in England in 1976.<sup>188</sup> At autopsy, lung tissue was

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<sup>185</sup> See Peto, Seidman, and Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," pp. 130-131, where the authors reached a similar conclusion and suggested that the rigidity of the amphiboles may be a necessary prerequisite for migration to the peritoneum.

<sup>186</sup> Curiously, the one mesothelioma at Charleston, South Carolina, was peritoneal.

<sup>187</sup> See Peto, Seidman, and Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," p. 134, where the authors suggested that these different ratios likely reflect differences in fibre dimension rather than chemical structure.

<sup>188</sup> J.S.P. Jones et al., "The Pathology and Mineral Content of Lungs in Cases of Mesothelioma in the United Kingdom in 1976," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 187-199.

available for analysis from 86 of these cases and also from 56 matched controls. The authors found that amphibole fibres, both crocidolite and amosite, occurred more frequently and in greater amounts in the lungs of the mesothelioma cases than the controls. Chrysotile fibres, on the other hand, were not present in any greater number of the cases than in the controls, and indeed in 30 of the cases of mesothelioma there were no chrysotile fibres at all.<sup>189</sup> Similar findings have been reported by A.D. McDonald, J.C. McDonald, and Pooley in their study of the mineral fibre content of lung tissue from North American mesothelioma victims in 1972.<sup>190</sup> They examined 99 cases of mesothelioma and matched controls. As in the English study, roughly equal quantities of chrysotile fibres were found in the cases and the controls; by contrast, higher quantities of amosite were found in the cases than in the controls, and higher quantities of crocidolite were likewise found in the cases than in the controls. The results of this study are set out in Table 5.29. These results, as the authors have fairly pointed out, must be treated with some caution by reason of the likelihood that chrysotile fibres disappear over the course of time.<sup>191</sup> Still, if chrysotile were responsible for a significant proportion of the mesothelioma tumours in this series, one would expect higher counts of this fibre in the lung tissue of the cases than were found.

Thus, the cumulative effect of all of the epidemiological evidence — cohort studies, case-series reports, and case-control studies of fibre content of lung tissue — presents a demonstrably convincing case against the amphiboles in relation to the incidence of mesothelioma. However, the animal evidence appears to present a much different picture. And it is the results of experimental studies with animals that have played an important role for those experts and regulatory agencies that do not believe there are substantial differences between the health hazards posed by chrysotile exposure, on the one hand, and amphibole exposure, on the other.<sup>192</sup> Again, it is not simply the fact that all fibre types have been able to induce mesothelioma in rats, whether the animals were dosed by inhalation or injection. In virtually all of the animal studies, chrysotile has produced at least as many or more mesothelial tumours than either crocidolite or amosite. The studies that have been carried out are far too numerous to permit us to report on all of them in detail. We choose to cite here the findings of a few of the experiments which we believe to be representative of the results of the vast majority of the studies.

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<sup>189</sup> *Ibid.*, p. 198.

<sup>190</sup> Alison D. McDonald, J. Corbett McDonald, and Fred D. Pooley, "Mineral Fibre Content of Lung in Mesothelial Tumours in North America," *Annals of Occupational Hygiene* 26:1-4 (1982): 417-422.

<sup>191</sup> RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 35-36; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 100.

<sup>192</sup> See, for example, RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, pp. 122-124.

Table 5.29  
Number (in Millions) of Asbestos Fibres per Gram of Dried Lung Tissue in 76 Male  
and 23 Female Cases of Mesothelioma in North America and Matched Controls

Number of Fibres	Chrysotile		Amosite		Crocidolite		Anthophyllite		Tremolite	
	Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls
Males										
None	3	1	37	47	47	60	58	61	29	31
<1	8	11	13	21	14	11	11	12	33	28
1<10	38	38	14	7	6	5	4	3	10	16
10<100	25	22	8	1	8	—	2	—	3	1
100<1,000	2	2	3	—	1	—	—	—	—	—
1,000 and over	—	2	1	—	—	—	—	—	1	—
Females										
None	—	—	17	15	15	16	18	17	7	9
<1	—	5	4	7	6	6	2	5	9	8
1<10	12	11	2	1	2	1	3	1	7	6
10<100	10	7	—	—	—	—	—	—	—	—
100 and over	1	—	—	—	—	—	—	—	—	—

SOURCE: Alison D. McDonald, J. Corbett McDonald, and Fred D. Pooley, "Mineral Fibre Content of Lung in Mesothelial Tumours in North America," *Annals of Occupational Hygiene* 26:1-4 (1982): 418 (Table 1).

In an early intrapleural injection experiment reported by Wagner and Berry in 1969, mesothelioma developed in 61 of the 96 rats given chrysotile, 55 of the 94 given crocidolite, and in only 38 of the 96 given amosite.<sup>193</sup> In an experiment reported by Reeves et al. in 1971, no mesotheliomas were produced by inhalation. But when the rats were injected with different types of asbestos, chrysotile produced 5 mesotheliomas; crocidolite, 4; and amosite, none.<sup>194</sup> A large 1974 inhalation experiment by Wagner et al. resulted in one mesothelioma (among 146 rats) from amosite and 4 mesotheliomas each from crocidolite (141 rats) and Canadian chrysotile (137 rats), although none from Rhodesian crocidolite (144 rats).<sup>195</sup> (See Table 5.25 in this chapter.) And in an often-cited intrapleural injection study, reported on by Wagner, Berry, and Skidmore in 1974, mesothelioma developed in 66% of the rats implanted with a superfine Canadian chrysotile, 61% implanted with UICC crocidolite, 36% implanted with UICC amosite, 30% implanted with UICC Canadian chrysotile, and 19% implanted with Rhodesian chrysotile.<sup>196</sup>

<sup>193</sup> J. Christopher Wagner and Geoffrey Berry, "Mesotheliomas in Rats Following Inoculation with Asbestos," *British Journal of Cancer* 23 (1969): 567-581.

<sup>194</sup> See Andrew L. Reeves et al., "Experimental Asbestos Carcinogenesis," *Environmental Research* 4 (1971): 496-511. The mortality and malignancy incidence of the rats exposed to asbestos in the experiment was as follows:

Species	Route	Amosite					Crocidolite					Chrysotile				
		Inventory				Incidence of Malignancy	Inventory				Incidence of Malignancy	Inventory				Incidence of Malignancy
		0	6	12	24		0	6	12	24		0	6	12	24	
		Months					Months					Months				
Rats	Inhalation	77	67	63	42	—	69	61	61	31	2 pulmonary cancers* both at 29 months	60	49	49	40	—
	Intratracheal	16	10	6	1	—	13	6	3	1	—	15	8	6	0	—
	Intrapleural	15	6	4	0	—	13	5	2	0	1 pleural mesothelioma** at 16 months.	12	6	4	1	2 pleural mesotheliomas** at 9 and 14 months.
	Intraperitoneal	11	3	0	0	—	13	7	4	0	3 peritoneal mesotheliomas*** at 12, 13, and 17 months.	13	7	0	0	3 peritoneal mesotheliomas*** at 7, 9, and 11 months.

Notes: \*Pulmonary carcinoma (squamous cell pattern).

\*\*Pleural mesothelioma (fibrosarcoma pattern).

\*\*\*Peritoneal mesothelioma (fibrous, papillary, osteogenic, or epithelial patterns)

SOURCE: Andrew L. Reeves et al., "Experimental Asbestos Carcinogenesis," *Environmental Research* 4 (1971): 502 (Table 4, excerpt)

See also, Andrew L. Reeves, Henry E. Puro, and Ralph G. Smith, "Inhalation Carcinogenesis from Various Forms of Asbestos," *Environmental Research* 8 (1974): 178-202.

<sup>195</sup> Wagner et al., "The Effects of the Inhalation of Asbestos in Rats."

<sup>196</sup> J. Christopher Wagner, Geoffrey Berry, and Joseph W. Skidmore, "Studies of the Carcinogenic Effects of Fiber Glass of Different Diameters Following Intrapleural Inoculation in Experimental Animals," in *Occupational Exposure to Fibrous Glass: A Symposium*, College Park, Maryland: 26-27 June 1974, HEW Publication no. (NIOSH) 76-151 (Washington, D.C.: U.S. Department of Health, Education and Welfare, April 1976), Table 18-3, p. 196. See also, World Health Organization, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, Table 17, p. 47.



The animal evidence, therefore, in striking contrast to the epidemiological evidence, appears to afford no basis for suggesting that chrysotile is any less hazardous in relation to the production of mesothelioma than the amphiboles. And the thrust of this animal evidence is consistent with the various *in vitro* or cell culture studies that have been reported, most of which indicate that chrysotile is at least as cytotoxic and haemolytic as the amphiboles.<sup>197</sup> In short, the human evidence and the experimental evidence appear to be quite inconsistent. We shall address this inconsistency later in this chapter.

### C.5 The Evidence in Relation to Gastrointestinal Cancer

As we have already observed, the association between asbestos exposure and cancer of the gastrointestinal tract appears to be less consistent and less marked when it has occurred among various cohorts of asbestos workers that have been studied than the association between asbestos and lung cancer, mesothelioma, or asbestosis.<sup>198</sup> However, when the incidence of gastrointestinal cancer is viewed in terms of fibre type, a somewhat clearer pattern emerges, although the type of exposure does not appear to us to explain completely the observed results. Our review of the available evidence does suggest that the finding of a dose-response relationship for gastrointestinal cancer and the existence of elevated mortality rates most frequently occur among amphibole-exposed populations. The incidence among the amosite factory workers at Paterson, the North American insulators, and the Belfast shipyard workers is demonstrably high;<sup>199</sup> and there have been excess deaths from abdominal cancers among a number of other industrial cohorts — the Scarborough cement workers, the Pennsylvania textile workers, the Manville retirees, and the East London factory workers<sup>200</sup> — exposed to mixtures of chrysotile and amphiboles. Still, there was no excess among the gas mask workers<sup>201</sup> or among the shipyard workers studied by Rossiter and Coles. Moreover, solely chrysotile-exposed populations have produced an excess of tumours from sites in the gastrointestinal tract. The SMR for abdominal cancer at Charleston was slightly

<sup>197</sup> Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, p. 17.

<sup>198</sup> See Chapter 2, Section D.4 of this Report.

<sup>199</sup> J.C. McDonald, "Asbestos-Related Disease: An Epidemiological Review," p. 594.

<sup>200</sup> In their latest report on the Manville retirees, Henderson and Enterline reported an SMR for gastrointestinal cancer of 137.8. Among the East London factory workers, there was a statistically significant excess of gastrointestinal cancer only among those workers most heavily exposed. (And, it may be noted, the authors included the peritoneal mesotheliomas with the gastrointestinal tumours.) For actual data on the relative risk of gastrointestinal cancer and the various cohorts discussed in this section, see Section B of this chapter.

<sup>201</sup> Jones et al., "The Consequences of Exposure to Asbestos in a Wartime Gas-Mask Factory," pp. 644–645. Among the women workers in the English gas mask factory, there were 10 cases of gastrointestinal cancer as against 20.3 expected.

elevated in Dr. Dement's cohort and even more so in Dr. A.D. McDonald's larger cohort; and even among the Quebec miners there was a substantial excess of cancer of the upper gastrointestinal tract among the most heavily exposed employees (although there was no excess at all in the cohort of cancer of the lower gastrointestinal tract).

On the other hand, there was no excess of gastrointestinal tumours at the Ferodo plant, none at the Cardiff asbestos-cement factory, and none among the Italian miners. There was no consistent dose-response relationship at the Connecticut friction products plant, and no excess of gastrointestinal tumours at Rochdale where, at least according to Mr. Peto, crocidolite exposure was minimal.<sup>202</sup> The cumulative effect of this evidence at least suggests, albeit not strongly, that an excess of gastrointestinal cancer is more likely to occur where there is amphibole exposure either alone or in conjunction with chrysotile than it is with chrysotile alone. This suggestion is consistent with the observed fact that there is a virtual absence of abdominal or peritoneal mesotheliomas among workers exposed only to chrysotile.

## C.6 The Evidence in Relation to Crocidolite and Amosite

Thus far we have focused our discussion on the differences, if any, between the health hazards of chrysotile exposure, on the one hand, and amphibole exposure, on the other. We have not discussed whether there is any evidence which might suggest that the health hazards of crocidolite and amosite are different. It is to this subject that we now turn.

It is generally thought that as between the two fibre types, crocidolite poses a greater danger to health than amosite, and this view is indeed reflected in the present Ontario Regulation Respecting Asbestos. There is a considerable amount of evidence that argues in support of that view. There appears to be a very high incidence of mesothelioma in crocidolite mining and a very low incidence in amosite mining; the considerable number of mesotheliomas observed among various cohorts of shipyard workers has been largely attributed to crocidolite exposure;<sup>203</sup> the crocidolite-exposed gas mask workers and Dr. Finkelstein's cohort of Scarborough cement

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<sup>202</sup> J.C. McDonald, "Asbestos-Related Disease: An Epidemiological Review," p. 594. See also, note 57, *supra*.

<sup>203</sup> J.C. McDonald, "Asbestos-Related Disease: An Epidemiological Review," p. 592. See also, Harries, "Experience with Asbestos Disease and Its Control in Great Britain's Naval Dockyards," p. 261. Amosite asbestos was also used in ship construction and may also be implicated in the relatively high incidence of mesothelioma among various shipyard workers. This may be particularly true in the United States where arguably amosite was more widely used than crocidolite in shipyards.

workers had a remarkably high disease incidence; and in animal experiments, crocidolite-exposed (and, for that matter, chrysotile-exposed) rats have invariably had a far worse record of tumour production than rats exposed to amosite.

Weighed against this evidence is the health experience of the North American insulators and the makers of insulation products. Admittedly the cohort at Paterson, New Jersey, had certain unusual features associated with it, but the fact remains that these amosite insulation workers had a particularly bad disease record, as we have previously discussed. The large cohort of North American insulators followed by Dr. Selikoff similarly had a high incidence of asbestos-related disease. The substantial number of mesotheliomas at the Pennsylvania textile factory arguably was attributable largely to the extensive use of amosite at that plant to make insulation products for the United States Navy. That part of the Henderson and Enterline cohort of retirees exposed solely to amosite, while admittedly small in number, had an excess incidence of lung cancer which closely approximated that part of the cohort exposed to crocidolite.<sup>204</sup> And lung tissue studies appear to support the proposition that amosite has played a significant role in the incidence of mesothelioma in North America.<sup>205</sup>

What is clear is that both amosite and crocidolite have resulted in serious disease records where utilized. If it is difficult to draw any definitive conclusions about the relative health hazards of amosite and crocidolite, this is because there are fewer studies of amosite-exposed populations than of crocidolite-exposed populations and also because there is an absence of actual exposure information in two principal cohorts where amosite has been used (the Paterson plant and the North American insulators). However, with respect to the latter deficiency, it is possible to derive exposure estimates, and we rely on such derived estimates in our risk assessment in Chapter 7. This assessment sustains the possibility that crocidolite is somewhat more hazardous than amosite. That possibility is further strengthened by evidence addressed in Section D of the present chapter. On balance, we conclude that the available evidence indeed suggests that crocidolite is somewhat more hazardous than amosite.

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<sup>204</sup> Henderson and Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," p. 122. Of the 1,075 retirees in the cohort, 58 were exposed to amosite only. There were 4 deaths from lung cancer and an SMR of 363.6.

<sup>205</sup> Alison D. McDonald, "Mineral Fibre Content of Lung in Mesothelial Tumours: Preliminary Report," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 681-685. See also, Table 5.29 in this chapter.



### C.7 The Evidence in Relation to Anthophyllite, Tremolite, and Actinolite

The health evidence on these three fibre types is scanty. There is one cohort study of which we are aware on the health risks of anthophyllite: that of Meurman, Kiviluoto, and Hakama, who investigated the mortality experience of the Finnish anthophyllite miners.<sup>206</sup> They reported an overall lung cancer SMR of 167, rising to 333 in those with more than 10 years' exposure, but no cases of mesothelioma and no excess cancer of the digestive system. Neither actinolite nor tremolite has been used in sufficient quantities commercially to engage detailed investigation of their health effects. However, as we have already pointed out, tremolite is known to be present in certain deposits of chrysotile asbestos and talc.<sup>207</sup> Rowlands, Gibbs, and A.D. McDonald have recently reported on their investigation of the mineral fibre content of 47 lung samples from the Quebec miners and millers (none, however, being a victim of either asbestosis or mesothelioma). Their striking finding was that tremolite was found in approximately similar quantities to chrysotile in these lung samples.<sup>208</sup> Large quantities of tremolite were also found in a series of 20 lung samples previously examined by Pooley from the same cohort.<sup>209</sup> And Dr. J.C. McDonald, during his testimony, went so far as to speculate that the mesothelioma among the Quebec miners heretofore attributed to chrysotile may be due to tremolite exposure.<sup>210</sup> Kleinfeld, Messite, and Zaki have investigated the mortality experience of a group of 250 miners of a talc deposit contaminated by tremolite, and to a

<sup>206</sup>L.O. Meurman, R. Kiviluoto, and M. Hakama, "Mortality and Morbidity Among the Working Population of Anthophyllite Asbestos Miners in Finland," *British Journal of Industrial Medicine* 31:2 (April 1974): 105-112. The authors' study cohort consisted of 1,092 miners first employed between 1936 and 1969, of whom 95% were traced and 248 had died.

<sup>207</sup>See Chapter 2, Section B.4 of this Report.

<sup>208</sup>Neil Rowlands, Graham W. Gibbs, and Alison D. McDonald, "Asbestos Fibres in the Lungs of Chrysotile Miners and Millers — A Preliminary Report," *Annals of Occupational Hygiene* 26:1-4 (1982): 411-417.

<sup>209</sup>Fred D. Pooley, "An Examination of the Fibrous Mineral Content of Asbestos Lung Tissue from the Canadian Chrysotile Mining Industry," *Environmental Research* 12 (1976): 281-298.

<sup>210</sup>RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 97-98. Dr. Davis testified that he had just completed an inhalation study with pure tremolite. While the study had been completed at the time of his testimony in January 1982, not all of the data had been analyzed. However, Dr. Davis suggested that his experiment appeared to indicate that tremolite is very harmful to rats and may be more harmful than chrysotile. See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 96-97. Mr. Ross Hunt testified that tremolite may be every bit as much a concern as crocidolite and noted that in 3 cases of mesothelioma in Cyprus, tremolite was the only asbestos fibre found by Dr. Fred D. Pooley at autopsy. See RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, p. 31.



lesser extent by anthophyllite, located in upper New York State.<sup>211</sup> The authors reported increased lung cancer mortality and one death from peritoneal mesothelioma. While this evidence indicates a possible health risk from tremolite exposure, in our judgement there are not yet sufficient data to come to any firm conclusion. However, we do point out that to the extent that tremolite contaminates mines in Ontario, and to the extent that it is utilized commercially, its health effects warrant further scientific investigation.

## C.8 Summary

In the first section of this chapter we observe that the incidence of asbestos-related disease appears to vary among industrial processes. In this section we observe that, at least in respect of the incidence of mesothelioma, the epidemiological evidence indicates there is a marked difference between chrysotile and amphibole exposure. It is, we believe, also fair to suggest that while chrysotile alone is capable of producing a high incidence of disease, as at Charleston, there are some processes in which it appears to have been used with little health risk — principally friction materials and to a lesser extent mining. On the other hand, there is no cohort systematically studied of which we are aware which has been exposed to amphiboles alone or to amphiboles in combination with chrysotile that has not demonstrated a considerable excess mortality associated with asbestos exposure. And yet, we also observe that the experimental evidence produces a conclusion that is quite contrary to the epidemiological evidence. Not only do all fibre types produce all asbestos-related diseases in animals, no matter what type of experiment has been undertaken, but also chrysotile appears to be no less hazardous than amosite or crocidolite, and this includes the production of mesothelioma as well as lung cancer and fibrosis.

How then do we account for these differences? Why is it that some industrial processes appear to be less hazardous than others? Why do the amphiboles appear to be largely responsible for the incidence of mesothelioma in humans? Why do the experimental evidence and the epidemiological evidence differ so strikingly in relation to the incidence of mesothelioma? It is to these questions that we now turn.

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<sup>211</sup>Morris Kleinfeld, Jacqueline Messite, and Mahfouz H. Zaki, "Mortality Experiences Among Talc Workers: A Follow-up Study," *Journal of Occupational Medicine* 16 (1974): 345-349. See also, John M. Dement and Ralph D. Zumwalde, "Occupational Exposures to Talc Containing Asbestiform Minerals," in *Dusts and Disease*, pp. 287-305.

## D. Reconciling the Evidence — Fibre Dimension and Other Considerations

The questions which we pose appear to beg a further question: What characteristics of asbestos fibres determine their pathogenicity? In our judgement, the starting point is the recognition of the significance of the dimensions of the fibre — its length, its diameter, its shape — in causing disease. In Chapter 4 we have already indicated that the diameter of the fibre plays an important role in its ability to become airborne, and therefore its ability to be inhaled. The thinner the diameter of fibres, the more likely they are to create respirable dust clouds. The diameter of the fibre is also important in terms of its ability to enter the respiratory tract and to penetrate into the lung, the pleural and even the abdominal cavities. Here too, the thinner the fibre, the more easily it can penetrate. Shape affects the behaviour of fibres because fibres that are curly and not straight behave as if they are thicker than their actual fibre diameter would suggest. Length is important in terms of the respirability of the fibre; but even more significantly, once the fibre has been inhaled, it is important in terms of one of the body's principal defence mechanisms: the activity of the macrophages. Longer fibres, that is, fibres with a minimum length of at least 5 or perhaps 8 microns, are not readily engulfed and immobilized by macrophages, and this may be a factor in their causing disease. To put the point differently, there is a preferential clearance of shorter fibres from the body.

These considerations taken together strongly suggest that it is the long, thin asbestos fibres that are apt to be most pathogenic. The late Dr. Mearl F. Stanton and his colleagues at the National Cancer Institute in the United States came to exactly this conclusion in a pathbreaking series of animal experiments which investigated the relationship between fibre dimension and carcinogenicity.<sup>212</sup> Dr. Stanton and his colleagues implanted various durable fibres of different but respirable size on the pleura of rats. They found that the biologic activity of these fibres in relation to the incidence of mesothelioma was very much dependent upon their dimension. In the most recent of their studies, reported in 1981, their results indicated that the probability

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<sup>212</sup> Mearl F. Stanton and Constance Wrench, "Mechanisms of Mesothelioma Induction with Asbestos and Fibrous Glass," *Journal of the National Cancer Institute (JNCI)* 48:3 (March 1972): 797-821; Mearl F. Stanton et al., "Carcinogenicity of Fibrous Glass: Pleural Response in the Rat in Relation to Fiber Dimension," *Journal of the National Cancer Institute* 58 (1977): 587-603; Mearl F. Stanton and Maxwell Layard, "The Carcinogenicity of Fibrous Minerals," in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, Gaithersburg, Maryland: 18-20 July 1977, NBS Special Publication 506 (Washington, D.C.: U.S. National Bureau of Standards, November 1978), pp. 143-150; and Mearl F. Stanton et al., "Relation of Particle Dimension to Carcinogenicity in Amphibole Asbestos and Other Fibrous Minerals," *Journal of the National Cancer Institute* 67:5 (November 1981): 965-975.

of mesothelioma correlated best with fibres 0.25 microns or less in diameter and 8 microns or more in length, but that a relatively high correlation was obtained for fibres of a dimension up to 1.5 microns in diameter and greater than 4 microns in length.<sup>213</sup> (See Table 5.30.) Experimental work by Dr. Friedrich Pott has also demonstrated that it is the long, thin fibres which are most pathogenic.<sup>214</sup> Thus, physics, biology, and experimental work all point to the significance of fibre dimension in causing asbestos-related disease, both in terms of the respirability of fibres and, once inhaled, in terms of their ability to reach the target site and to cause disease.

And when consideration is given to fibre dimension in assessing the epidemiological and animal evidence, a more consistent and coherent picture begins to emerge. The amphiboles generally, and crocidolite in particular with its straight, thin, needle-like dimensions, have a greater ability to become airborne than chrysotile and therefore a greater ability to create a respirable dust cloud than chrysotile.<sup>215</sup> To use a common phrase, the amphiboles, and especially crocidolite, are dustier than chrysotile. As between crocidolite and chrysotile, experiments have demonstrated that chrysotile requires much more active disturbance to generate a dust cloud than does

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<sup>213</sup>Stanton et al., "Relation of Particle Dimension to Carcinogenicity in Amphibole Asbestos and Other Fibrous Minerals," pp. 965, 973. In their earlier work, Dr. Stanton and his colleagues concluded that the most carcinogenic fibres were those longer than 8 microns and thinner in diameter than 1.5 microns. See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, p. 43. Dr. Davis testified that the notion that a long fibre might be more hazardous than a short fibre goes back as far as the work of Professor E.J. King in 1946. See E.J. King, J.W. Clegg, and V.M. Rae, "Effect of Asbestos, and of Asbestos and Aluminium, on the Lungs of Rabbits," *Thorax* 1 (September 1946): 188-197.

<sup>214</sup>Friedrich Pott, "Some Aspects on the Dosimetry of the Carcinogenic Potency of Asbestos and Other Fibrous Dusts," *Staub-Reinhalt. Luft* 38:12 (December 1978): 486-490. According to Dr. Eric J. Chatfield, while Dr. Pott suggested a declining carcinogenicity factor below 5 microns in length, he still indicated that fibres greater than 1 micron in length would have an effect. Dr. Chatfield observed that if Dr. Pott is correct, the overall effect of short fibres may be larger than the effect of longer fibres simply because there are more of them in any dust cloud. See RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 14-15. See also, Ontario, Royal Commission on Asbestos, Exhibit II-27 [hereafter RCA Exhibit], Tab 19, in RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18: Eric J. Chatfield, "Airborne Asbestos Fibres: A Summary of Some Measurement Problems," Sheridan Park, Ontario Research Foundation, 1981, p. 1. (Mimeographed.)

<sup>215</sup>See RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 96; RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, p. 28; RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 94; and RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 11.

Table 5.30

**Correlation Coefficients of Logit of Tumour Probability  
with Common Logarithm of Number of Particles per Microgram  
in Different Dimensional Ranges**

Fibre Diameter (Microns)	Fibre Length (Microns)		
	≤4	>4-8	>8
>4	—	-0.28	-0.30
>1.5-4	-0.45	-0.24	0.13
>0.25-1.5	0.01	0.45	0.68
≤0.25	0.20	0.63	0.80

SOURCE: Mearl F. Stanton et al., "Relation of Particle Dimension to Carcinogenicity in Amphibole Asbestoses and Other Fibrous Minerals," *Journal of the National Cancer Institute (JNCI)* 67:5 (November 1981): 973 (Table 3).



crocidolite.<sup>216</sup> Further, these experiments have also indicated that crocidolite will repeatedly become airborne, whereas when chrysotile is allowed to settle out on any horizontal or vertical surface, it is much less likely to become airborne again. Also, when water is applied to chrysotile it appears to prevent the fibres from becoming airborne, whereas crocidolite will dry out and become respirable again.<sup>217</sup>

Not only are crocidolite fibres more likely to become airborne and hence respirable in any given dust cloud, crocidolite is more likely to have a higher percentage of fibres of hazardous dimension than chrysotile or indeed, it may be added, than amosite.<sup>218</sup> To put this point in practical terms, it would appear that at any given fibre concentration as measured by the optical microscope, there are more likely to be a larger number of long, thin fibres in a crocidolite dust cloud than in either an amosite or chrysotile dust cloud. As between the latter two fibre types, the evidence suggests that amosite is more likely to generate a greater number of fibres of hazardous dimension than chrysotile.

Once inhaled, the needle-like shape of the amphiboles also likely allows for deeper penetration into the peripheral portions of the lung, pleural, and abdominal surfaces than chrysotile fibres which, because of their curliness, have a greater cross-sectional area, thereby increasing the

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<sup>216</sup>RCA Exhibit II-57, Tab 8, in RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41: Ross Hunt, "The Asbestos Bundle Cleavage Difference Between Sheet Silicate (Chrysotile) and the Chain Silicate (Crocidolite)," Cleckheaton, Yorkshire, BBA Group PLC, Industrial Health Unit, 26 May 1982. (Mimeographed.) See also, W.H. Walton, "The Nature, Hazards and Assessment of Occupational Exposure to Airborne Asbestos Dusts: A Review," *Annals of Occupational Hygiene* 25 (1982): 117-247, as cited in Acheson and Gardner, *Asbestos: The Control Limit for Asbestos*, paragraph 37, p. 7; and Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, pp. 17-18.

<sup>217</sup>RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, pp. 75-77, 84.

<sup>218</sup>RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 96. Mr. Berry testified that the crocidolite fibres that tend to be distributed in the air are finer than the chrysotile fibres. Further, the long fibres of chrysotile are not completely straight, which means that aerodynamically they behave as if they are a lot thicker than the actual fibre diameter would suggest. See also, Walton, "The Nature, Hazards and Assessment of Occupational Exposure to Airborne Asbestos Dusts: A Review"; Chung-Yung Hwang and Graham W. Gibbs, "The Dimensions of Airborne Asbestos Fibres — I. Crocidolite from Kuruman Area, Cape Province, South Africa," *Annals of Occupational Hygiene* 24:1 (1981): 39; and Graham W. Gibbs and Chung-Yung Hwang, "Dimensions of Airborne Asbestos Fibres," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 69-78.

opportunity for interception, decreasing the likelihood of penetration, and possibly decreasing movement after tissue penetration.<sup>219</sup>

These considerations provide a plausible explanation for the greater incidence of mesothelioma and for the fact that peritoneal mesothelioma occurs almost exclusively among amphibole-exposed populations; and also suggest that on balance the amphiboles are likely to be more hazardous than chrysotile. As between the amphiboles, in general crocidolite is likely to be somewhat more hazardous than amosite. Overall, there appears to be a correlation between fibre type and fibre dimension and thus at least to some degree a correlation between fibre type and the incidence of asbestos-related disease.

But precisely because the dimension of the fibre affects the incidence of disease, any asbestos fibres in the critical size range, no matter what the type, are likely to be hazardous. In other words, while the amphiboles may be more likely to generate airborne fibres of hazardous dimension, to the extent that particular industrial processes can generate finer or thinner chrysotile fibres, we would expect the hazardous nature of chrysotile exposure to increase. And that appears to have taken place. For when account is taken of the dimension of the fibre used in industry, the differences in the incidence of disease between mining and manufacturing, and, within manufacturing, as between textiles and friction products, become more understandable. For example, it is now widely thought that long, thin fibres are far more prevalent in textile operations than in other kinds of industrial or mining operations. The vigorous activity involved in certain of the textile operations, especially spinning and carding, tends to cause the fibres to split longitudinally, thus increasing the absolute number of fibres and making each thinner in diameter.<sup>220</sup> Chrysotile, which has a greater tendency to separate into fibrils than amphiboles, is likely to be particularly affected in this manner by textile operations.<sup>221</sup> In the manufacture of friction products nothing of this kind appears to take place. Rather, the fibres are

<sup>219</sup> Peto, Seidman, and Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," p. 134; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 45; Paul Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, p. 136; R.L. Zielhuis, rapporteur, *Public Health Risks of Exposure to Asbestos*, Report of a Working Group of Experts, prepared for the Commission of the European Communities (Oxford: Pergamon Press, 1977), p. 10.

<sup>220</sup> RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, pp. 46, 112; RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 37; RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 41-42; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 58.

<sup>221</sup> RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 41-42. Dr. A.D. McDonald testified that the carding and weaving of chrysotile textiles fragment chrysotile as much as that fibre can be fragmented.

ground together, causing them to break horizontally, thereby making them shorter in length but not changing their diameter.<sup>222</sup>

In fact, Dr. Dement and his colleagues investigated the characteristics of the airborne fibres at Charleston, using transmission electron microscopy, and found that 10 to 27% of all airborne fibres were longer than 5 microns in length. Moreover, compared with friction product and cement product operations using chrysotile, textiles were found to have a substantially higher fraction of the long, thin fibres that Stanton and others have implicated as being most carcinogenic.<sup>223</sup> A similar finding was made by Dr. Graham W. Gibbs who testified before us that the proportion of long fibres in certain areas of textile manufacturing, like spinning, appears to be substantially more than in mining. He noted that the mining industry produces a whole variety of different grades of fibres. However, in the secondary industries such as textiles, it is only longer fibres that appear to be utilized.<sup>224</sup> In a subsequent study of the dimension of different fibres in a variety of industrial processes, Gibbs and Hwang have shown that in terms of diameter alone, chrysotile fibres used in textile carding operations are potentially more respirable than chrysotile or other fibres at different stages of processing.<sup>225</sup>

These findings assist in explaining the otherwise startling difference in disease incidence between the textile factory at Charleston, on the one hand, and, on the other, the friction plants at Derbyshire (the Ferodo plant) and Connecticut and the asbestos-cement factory at Cardiff. These findings, however, also clearly demonstrate that, subjected to certain processing, chrysotile is quite capable of producing fibres of very hazardous dimension.

A consideration of the physical parameters of asbestos fibres also appears to reconcile the observed incidence of mesothelioma in and around the Cape crocidolite mines in South Africa and the apparent absence of this

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<sup>222</sup> Zielhuis, *Public Health Risks of Exposure to Asbestos*, p. 22. Data compiled for this report indicated that the average fibre length in textile operations is between 8 and 24 mm; in asbestos-cement operations, between 2 and 13 mm; and in friction materials operations, between 2 and 4 mm. See also, RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 112; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 58.

<sup>223</sup> Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," p. 883. (Discussion.) Dr. Dement indicated that on the basis of a deposition model used in their investigation, it was estimated that 13 to 15% of the airborne particles longer than 5 microns in length would be deposited in deep, non-ciliated portions of the lung.

<sup>224</sup> RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 125.

<sup>225</sup> Graham W. Gibbs and Chung-Yung Hwang, "Physical Parameters of Airborne Asbestos Fibres in Various Work Environments — Preliminary Findings," *American Industrial Hygiene Association Journal* 36:6 (June 1975): 464.



tumour among the crocidolite and amosite miners of the Transvaal. Timbrell, Griffiths, and Pooley, in comparing the fibre dimensions from these two regions, ascertained that: (i) crocidolite fibres from the Cape are finer by a factor of 3; (ii) due to their larger diameter, the free-falling speed of Transvaal fibres is 9 times that of Cape fibres, meaning that Transvaal fibres are less likely to become airborne and will settle more rapidly; and (iii) fibres from the Transvaal have a greater aerodynamic size and thus a greater likelihood of interception in the larger airways of the tracheal-bronchial tree and less likelihood of penetrating to the periphery of the lungs.<sup>226</sup>

Further confirmation that the different health risks in mining are a function of fibre dimension comes from the work of Gibbs and Hwang who have compared the dimensions of fibres collected in the chrysotile mines in Quebec, the amosite mines of the Transvaal, and the crocidolite mines of the Cape.<sup>227</sup> Crocidolite fibres, because of their very thin diameter and relative length, tended to have a higher aspect ratio (length to diameter ratio) than either amosite or chrysotile. Amosite fibres, although thicker in diameter than the other two fibre types, were also longer and thus tended to occupy a middle position in terms of aspect ratio. Chrysotile fibres had the lowest aspect ratio of all. If it is assumed that fibres observed by optical microscopy are greater than 5 microns in length and greater than 0.3 microns in diameter, the percentage of total airborne fibres observed by Gibbs and Hwang during the bagging of crocidolite was 0.6%; of amosite, 16.9%; and of chrysotile, 1.6%. Conversely, if it is assumed that fibres greater than 5 microns in length and less than 0.3 microns in diameter are most hazardous, the number of fibres in that size range for concentrations of 2 f/cc, as measured by the optical microscope, would be 23 f/cc for crocidolite, compared to 0.9 f/cc for amosite, and 3 f/cc for chrysotile. Clearly, the results of this study in terms of fibre dimension are quite consistent with the observed incidence of mesothelioma among the three mining regions.<sup>228</sup>

The studies by Gibbs and Hwang also offer an explanation for the high incidence of disease among North American insulation workers exposed to amosite. While the percentage of long, thin fibres in dust clouds at the Transvaal mines appeared to be quite low and was thus consistent with

<sup>226</sup> V. Timbrell, D.M. Griffiths, and F.D. Pooley, "Possible Biological Importance of Fibre Diameters of South African Amphiboles," *Nature* 232 (2 July 1971): 55-56. The mean fibre diameters for the samples used by the authors were as follows: North Western Cape crocidolite, 0.073 microns; Transvaal crocidolite, 0.212 microns; and Transvaal amosite, 0.243 microns. The free-falling speed of fibres (a measure of particle aerodynamic size) is nearly proportional to the diameter squared and increases only slowly with an increase in length.

<sup>227</sup> Gibbs and Hwang, "Dimensions of Airborne Asbestos Fibres."

<sup>228</sup> *Ibid.*, p. 76.



the apparent absence of asbestos-related disease from these mines, the diameter of these fibres tended to decrease considerably from the raw amosite dumping stage to the stage when it was to be applied to building surfaces for insulation.<sup>229</sup> Indeed, the authors' results suggested that at this stage amosite dust concentrations had a slightly higher percentage of fibres greater than 5 microns in length and less than 0.5 microns in diameter than raw crocidolite and a much higher percentage of fibres of these dimensions than chrysotile in textile carding operations.<sup>230</sup>

What these observations all tend to indicate is that there is indeed an association between type of industrial process and fibre dimension, and because of this, there is accordingly an association between industrial process and disease. A consideration of fibre dimension thus serves to assist in explaining the differing disease incidence in various industrial cohorts and has served to assist in explaining the differing incidence at least in mesothelioma among asbestos workers exposed to different fibre types. In our judgement, a consideration of fibre dimension also serves to reconcile the otherwise conflicting animal and epidemiological evidence on fibre type.

The most successful experiments in producing malignant tumours have been injection experiments. But these experiments do not take account of the importance of fibre dimension in terms of the respirability of the fibres and, once inhaled, in terms of their capacity to reach the site of tumour production. Moreover, most animal experiments have tended to use fibres of dimensions not found in industry. The UICC Reference Samples, including those for chrysotile, are all much finer in diameter than are those used industrially.<sup>231</sup> When the size distribution of the fibres used in animal experiments is taken into account, then the animal data and human epidemiological data become quite consistent. For example, in the large animal injection experiment reported on by Wagner, Berry, and Timbrell in 1973, UICC crocidolite with a higher proportion of long fibres produced

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<sup>229</sup>Gibbs and Hwang, "Physical Parameters of Airborne Asbestos Fibres in Various Work Environments — Preliminary Findings," p. 464.

<sup>230</sup>*Ibid.*, p. 465. The authors' study provided the following percentages of fibres with lengths greater than 5 microns and diameters less than 0.5 microns present in airborne dust: amosite application of insulation, 18.3%; crocidolite, 17.5%; amosite dumping, 17.0%; chrysotile dryer, 1.45%; chrysotile bagging, 1.0%; and chrysotile carding, 0.4%. The authors noted that if the combination of length greater than 5 microns and diameter less than 0.5 microns was the only factor responsible for the production of mesothelioma, their results would suggest that the hazard for the manufacturer or the insulation worker using amosite would be considerably greater than for chrysotile workers. *Ibid.*

<sup>231</sup>RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 29, 76–77. See also, Wagner et al., "The Effects of the Inhalation of Asbestos in Rats," p. 268; the authors observed, however, that there appears to be a trend for industry to use finer chrysotile, so that experimental results may be more relevant to current industry than to past.

more mesotheliomas than the UICC chrysotile.<sup>232</sup> Only the artificially separated superfine Canadian chrysotile appeared to be more hazardous. The recent inhalation study by Dr. Davis, in which chrysotile produced more fibrosis than the amphiboles and produced all of the malignant lung tumours, is readily explained as a matter of fibre dimension: 7% of all fibres in the chrysotile dust clouds were 20 or more microns in length, whereas in the amphibole dust clouds less than 0.5% of all fibres were greater than 20 microns in length.<sup>233</sup> *In vitro* tests also support the proposition that only longer fibres have a pathogenic effect on cell membranes, with fibres less than 5 microns in length being quickly and completely engulfed.<sup>234</sup>

Thus, we conclude from all of this evidence that the pathogenicity of asbestos is primarily a function of fibre dimension. We also conclude that the amphiboles, and especially crocidolite, are more likely to be of hazardous dimension than chrysotile. Moreover, the significance of fibre dimension in the production of disease has implications for fibre substitutes for asbestos. To the extent that man-made mineral fibres are long and thin in dimension, their use may also pose a health risk.<sup>235</sup> The health effects of substitutes for asbestos are considered in more detail in Chapter 6, Section E.

But while we believe that fibre dimension is the most biologically relevant characteristic of asbestos, we cannot totally discount the importance of other considerations. One such consideration is the chemistry and surface properties of asbestos fibres. Several studies published in the late 1960s and early 1970s have pointed to the possible importance of the fibre's surface chemistry in causing disease, although there is some disagreement as to which of the chemical characteristics of asbestos fibres are important.<sup>236</sup> There is, however, some evidence that the chemistry of chrysotile, and particularly its magnesium component, is such that it is more likely to dissolve in lung fluid over time. It is known that the amphiboles generally will not

<sup>232</sup> J. Christopher Wagner, Geoffrey Berry, and Victor Timbrell, "Mesotheliomata in Rats after Inoculation with Asbestos and Other Materials," *British Journal of Cancer* 28:2 (August 1973): 173-185. See also, Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, p. 19.

<sup>233</sup> John M.G. Davis, "The Use of Animal Models for Studies on Asbestos Bioeffects," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 797.

<sup>234</sup> E.G. Beck, "Experimental Pathology — *In Vitro* Studies — Related to Asbestos and Other Mineral Fibres," in *Biological Effects of Mineral Fibres*, vol. 1, p. 389.

<sup>235</sup> For example, Stanton and Wrench found that glass fibres in the size range over 8 microns in length and less than 1.5 microns in diameter were almost as effective as asbestos in producing tumours after animal injection experiments. See Stanton and Wrench, "Mechanisms of Mesothelioma Induction with Asbestos and Fibrous Glass."

<sup>236</sup> Many of the studies suggesting the importance of the chemistry and surface properties of asbestos fibres are discussed in Jacques Dunnigan et al., "Cytotoxic and Haemolytic Effect of Native and Chemically Modified Chrysotile," *Fourth International Conference on Asbestos*, Turin, Italy: 26-30 May 1980, Preprints, vol. II (Turin, Italy: Istituto di Arte Mineraria del Politecnico & Associazione Mineraria Subalpina, 1980), pp. 747-772.

dissolve and thus may be likely to last longer in the lung than chrysotile.<sup>237</sup> And there is evidence to suggest that chrysotile fibres are cleared more readily from the lungs than are amphibole fibres.<sup>238</sup> If this were the case, it would provide an additional explanation for the apparently different results between the animal and human data insofar as chrysotile is concerned. It is possible that for all species, tumour induction requires a large fraction of a lifespan. It is possible that chrysotile may be able to survive in animals long enough to exert its full carcinogenic potential but may not always be able to do so in humans.<sup>239</sup>

A group of epidemiologists from France who have been studying the health effects of asbestos place particular importance on the surface properties of asbestos as a determinant of disease. While they clearly admit the importance of fibre dimension in explaining how asbestos fibres reach the site of tumours, they also believe that once at the site, the interaction between the surface of the fibre and the surface of the cell has significance in disease production.<sup>240</sup>

Dr. Jacques Dunnigan of the Institut de recherche et de développement sur l'amiante at Sherbrooke, Quebec, testified before us that there is evidence that asbestos fibres become chemically altered or coated during processing and that these alterations may have the effect of making fibres less toxic.<sup>241</sup> Two examples he gave were the encapsulation of chrysotile fibres in resin in certain asbestos applications such as the manufacture of

<sup>237</sup> RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 67. Dr. Davis testified that it can be demonstrated that even in a weak acidic solution the magnesium ions can be leached out of the structure of a chrysotile fibre. One is still left with a fibril that looks much the same, but chemically it can be shown to have much less magnesium. Once the magnesium has left, the fibril is physically much weaker and more easily broken up. See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 55-56.

<sup>238</sup> See Margaret R. Becklake, "Exposure to Asbestos and Human Disease," *The New England Journal of Medicine* 306:24 (17 June 1982): 1480-1482. The evidence that there is a preferential clearance of chrysotile as compared with amphibole fibres from the lung is largely based upon case-control lung tissue studies which indicate an excess of amphiboles in the lungs of the cases whereas the chrysotile content was similar in both cases and controls.

<sup>239</sup> Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, p. 17.

<sup>240</sup> Personal communication between Professor Patrick Sebastien, Institute of Occupational Health and Safety, McGill University, Montreal, Quebec and Royal Commission on Asbestos Staff, 21 April 1983. See also, J. Bignon and M.C. Jaurand, "Biological *In Vitro* and *In Vivo* Responses of Chrysotile Versus Amphiboles," *Environmental Health Perspectives* 51 (September 1983): 73-80.

<sup>241</sup> See RCA Transcript, Evidence of Dr. Jacques Dunnigan, 20 August 1981, Volume no. 28, pp. 43-51, 83-88.



friction products and the calcium-coating that may chemically change chrysotile during asbestos-cement manufacturing.<sup>242</sup> In a similar vein, it has been suggested by some authorities that the milling process destroys the fibrous structure of chrysotile to a greater extent than the amphiboles.<sup>243</sup> Dr. Dunnigan and others are now engaged in research which seeks to alter the chemical characteristics of asbestos so as to render the fibres less toxic but at the same time maintain their useful properties.

In our judgement, the chemistry and the surface properties of asbestos fibres have yet to be shown to be as important as fibre dimension in determining disease. However, in a subject that is bedevilled by uncertainty, we do not discount the fact that these characteristics of asbestos fibres may have a significant role to play. It may well be the case that future research will cast a clearer light on their importance. At this stage we draw sustenance from the fact that the evidence which does exist in the realm of chemistry and surface properties points to a similar conclusion as the evidence on fibre dimension: namely, that chrysotile tends to be less hazardous than the amphiboles.

There is one further and related consideration that also tends to suggest chrysotile may be less hazardous. This is its tendency to split apart continuously and ultimately to break up into tiny fibrils which might then be easily cleared from the lungs. Whether it is a matter of fibre chemistry or the physical tendency of chrysotile to split into tiny fibrils, there is considerable evidence that chrysotile fibres do not have the same long-term

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<sup>242</sup> Ibid., pp. 43-48, 83-84. In his testimony, Dr. Dunnigan did not suggest that fibre dimension should be disregarded in assessing the toxicity of asbestos fibres, but rather that the chemical properties of fibres should be considered in addition to fibre dimension. RCA Transcript, Evidence of Dr. Jacques Dunnigan, 20 August 1981, Volume no. 28, p. 64. In his testimony, Dr. Weill noted that it had been suggested that asbestos in the asbestos-cement industry has a calcium-containing coating and that this coating may make it less pathogenic. See RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 97.

<sup>243</sup> RCA Transcript, Evidence of Dr. Jacques Dunnigan, 20 August 1981, Volume no. 28, pp. 25-26. See also, K.R. Spurny, H. Opiela, and G. Weiss, "On the Milling and Ultrasonic Treatment of Fibres for Biological and Analytical Applications," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 931-933.



durability in the lungs as do the amphiboles.<sup>244</sup> This observation is consistent with the results of lung tissue studies which strongly suggest that there is a preferential clearance of chrysotile from the lungs as compared to the amphiboles.<sup>245</sup> To the extent that length of residence in the lung is important in disease production, this consideration too would implicate the amphiboles as more hazardous.

These various considerations that we have discussed, particularly fibre dimension, appear to offer a reasonable explanation for the observed incidence of asbestos-related disease. But, in our judgement, a degree of uncertainty still remains. For example, we cannot totally dismiss the importance of short fibres in the production of disease. While experimental studies indicate that, once in place, longer fibres are more carcinogenic than short, it has been suggested that shorter fibres more easily migrate through the body and penetrate the tissue and thus more easily reach the sites of tumours.<sup>246</sup> It has also been suggested that due to the much larger number of short fibres, their overall effect may be greater than long fibres depending upon the size distribution of a given dust cloud, although this suggestion appears to be inconsistent with the engulfing activity of the macro-

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<sup>244</sup>In his review article, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, at p. 7, Dr. Davis noted that the reason why most researchers have found relatively more amphibole fibres in human lung specimens, in spite of the fact that chrysotile makes up the bulk of industrial asbestos usage, was probably indicated by M.C. Jaurand et al. in 1976. In an article entitled "Solubilité du chrysotile *in vitro* et dans le poumon humain," *Revue française des Maladies Respiratoire* 4, Supp. 2 (1976): 111-120, the authors produced evidence of the chemical dissolution of chrysotile in the lung tissue. Dr. A.D. McDonald testified that amphibole fibres tend to remain as longer fibres once they get into body tissue, whereas chrysotile fibres tend to split up into much shorter pieces. See RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 47-48. See also, RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 131-132. In his testimony, Dr. Gibbs noted that amphibole fibres are very acid resistant and certainly not water soluble. Chrysotile, on the other hand, has been demonstrated to be not very acid resistant, and it has a magnesium hydroxide outer layer, so the possibility for leaching to occur does exist. But Dr. Gibbs also noted that the ability to break off a bundle is far greater for chrysotile than it is for amphibole fibres. This ability of chrysotile fibres to break apart might well improve their ability to be removed from the lung.

<sup>245</sup>See Becklake, "Exposure to Asbestos and Human Disease," and the lung tissue studies cited therein.

<sup>246</sup>See RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 47-48; RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 130; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, p. 71.

phages.<sup>247</sup> A recent study by Professor Patrick Sebastien et al. analyzed the asbestos fibre content of tissue of the pleura from 29 cases exposed to mixed asbestos dusts and with a variety of tumours.<sup>248</sup> The surprising finding of this study was that virtually all of the fibres retained in the pleura were short chrysotile fibres, whereas the longer amphibole fibres were found in the lung parenchyma tissue. We cannot account for this finding if it suggests that short chrysotile fibres are largely responsible for mesothelioma production, since it is a finding which is contrary both to experimental work on fibre dimension and to the known incidence of mesothelioma in human populations.<sup>249</sup>

There is other uncertainty. For example, it is not entirely clear in our judgement why chrysotile is capable of producing an extraordinarily high incidence of lung cancer and yet only one mesothelioma has been found at Charleston. It may be that chrysotile fibres, even in the hazardous fibre dimensions that are likely found in textile operations, are still not able to migrate to the pleura or to the peritoneal cavities as efficiently as the amphiboles, but this is not known with certainty.

There are different cohorts of workers manufacturing the same products, apparently using the same processes and the same mix of fibre types, that still exhibit widely different health effects. For example, there is a substantial difference in the amount of asbestos-related disease between the Johns-Manville plant at Scarborough and the two asbestos-cement plants at New Orleans, a difference that persists even when account is taken of the selective exposure to crocidolite at the New Orleans factory. Neither Dr. Finkelstein nor Dr. Weill has been able to offer a plausible explanation for

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<sup>247</sup>RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 47-48. Dr. Chatfield, in "Airborne Asbestos Fibres: A Summary of Some Measurement Problems," at p. 1, noted the work of Dr. Pott which indicated that although there was a declining carcinogenicity factor for a single fibre below 5 microns in length, it was still significant below this length. Dr. Chatfield went on to observe that since in airborne material there are many more short fibres than longer ones, the overall biological effect of the shorter fibres may actually be larger than that of the less numerous long fibres.

<sup>248</sup>Patrick Sebastien et al., "Asbestos Retention in Human Respiratory Tissues: Comparative Measurements in Lung Parenchyma and in Parietal Pleura," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 237-246.

<sup>249</sup>This somewhat surprising finding was referred to by a number of witnesses who testified at our hearings. See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 71-73; RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 130; and RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 126-127. In his testimony, Dr. Davis suggested the possibility that the cases studied by Sebastien et al. may have had long fibres of chrysotile in the pleura previously which had broken up over time. Another possibility is that the break-up could have taken place elsewhere within the body and that subsequently these now shorter fibres could have found their way to the pleura and remained there.

this difference.<sup>250</sup> It could be that the explanation might be found in climatic differences between Ontario and Louisiana and consequent differences in plant ventilation.

What this discussion amply illustrates is that the final chapter on the pathogenesis of asbestos-related disease is yet to be written. However, we believe that the conclusions that we have reached in this chapter fairly represent the current state of the evidence.

## E. Dose-Response Relationships

### E.1 The General Principle

The dose-response relationship is a general biological principle that refers to the correspondence between the extent of exposure (that is, the “dose”) and the biological outcome of interest (the “response”). If one can demonstrate an increasing (or decreasing) risk of severity of response, for example, lung cancer, in association with an increased (or decreased) dose of exposure to the putative cause, for example, asbestos, then one can say there is a dose-response relationship.<sup>251</sup> The nature of that relationship will turn upon the shape and location of the line or curve when it is plotted on a graph. If, for example, an increase in the dose of asbestos leads to a proportional increase in the excess of lung cancer, then the dose-response relationship for lung cancer is said to be linear in nature and is described by a straight line. Shapes other than linear are clearly possible.

The significance of the concept of a dose-response relationship is at least twofold. First, it is a key element in establishing a causal relationship between the dose and the response as well as a tool for explaining variations in biological response. Second, in practical terms, it provides information upon which to establish regulatory standards. A linear dose-response relationship is particularly attractive for regulatory purposes because, once assumed, it permits by extrapolation a calculation of relative risk at exposure levels too low to be detected by normal epidemiological studies.<sup>252</sup>

<sup>250</sup>Letter from Dr. Hans Weill, Science Policy Fellow, The Brookings Institution, Washington, D.C. to the Royal Commission on Asbestos, 13 July 1983.

<sup>251</sup>For a discussion of the dose-response relationship, see David L. Sackett, “A Review of Diagnostic Tests for Causation,” in Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos, Second Public Meeting, Friday, December 12, 1980*, reported by Lydia Dotto (Toronto: Royal Commission on Asbestos, 1981), pp. 13–14 and Appendix B, pp. 6–8.

<sup>252</sup>J.C. McDonald and Liddell, “Mortality in Canadian Miners and Millers Exposed to Chrysotile,” p. 8.

## **E.2 A Threshold or “No-Effect” Level**

Closely related to the concept of a dose-response relationship is the notion of a threshold: that is, a level of dose below which no effect or no risk of illness or death will occur. When one charts a dose-response relationship on a graph to ascertain whether or not a threshold exists, one should examine the location and contour of the dose-response relationship at the point of zero dose. If the response at the point of zero dose is zero (or positive), and the slope of the dose-response line or curve is positive (that is, a line going out from the origin), this suggests there is no dose at which some response will not occur. In other words, this suggests that no threshold exists. If, on the other hand, there is zero response at a dose greater than zero, this suggests that there are doses at which no change in response is expected; that is, a threshold or “no-effect” level exists.<sup>253</sup>

The existence or non-existence of a threshold for asbestos-related diseases has stirred considerable controversy among those who seek to legislate an acceptable level of exposure. If indeed a threshold level does exist, such a level poses no risk. If there is no threshold, then we must accept the proposition that whatever level of exposure is chosen as the regulatory control limit, it will always carry with it at least some risk of disease.

## **E.3 The Nature of the Dose-Response Relationships for Asbestos-Related Diseases**

From the foregoing it is evident that the nature of the dose-response relationships for the asbestos-related diseases and the existence or non-existence of a threshold or no-effect level for these diseases are important from a regulatory standpoint. For ease of exposition, we discuss dose-response in relation to the three principal diseases separately.

### **(a) Asbestosis**

The preponderance of data from the various working populations that have been studied — including the Quebec chrysotile miners, the Rochdale and Charleston textile workers, and the Scarborough asbestocement workers — clearly evidences a positive dose-response relationship for asbestosis.<sup>254</sup> And while we are told that biologically a linear dose-response relationship is less plausible for a chronic fibrotic process such as asbestosis

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<sup>253</sup>Sackett, “A Review of Diagnostic Tests for Causation,” Appendix B, p. 8.

<sup>254</sup>See Section B of this chapter.



than it is for cancer,<sup>255</sup> nevertheless the data are consistent with a linear relationship, and we are satisfied that such a linear relationship appears to describe the results of the various studies satisfactorily.<sup>256</sup>

We are less satisfied that there is no threshold for the clinical manifestation of asbestosis. Indeed, there was a general consensus among the expert witnesses who appeared before us that mortality associated with asbestosis is related to high doses and that at the current much lower exposure levels found in industry even clinically disabling asbestosis is much less a matter of concern than it has been in the past.<sup>257</sup> Biologically this seems plausible. Unlike cancer, which may be initiated by a "trigger dose" of asbestos, it would seem that the clinical manifestation of asbestosis requires the inhalation of a considerable amount of asbestos over time. As Dr. J. Corbett McDonald testified, a person has "... to have fibrosis beyond a certain level before it could conceivably do anything to you, because the reserve power of the lung is fairly substantial. . . ."<sup>258</sup>

Viewed in this light, what is material about asbestosis involves not so much the existence or non-existence of a threshold for the initiation of fibrosis; what is more relevant is the relationship of asbestos exposure to the development of the fibrotic process. As we assess the epidemiological literature there is a growing body of data to suggest, if not confirm, that there is a low level of asbestos exposure below which the clinical manifestation of asbestosis will not occur, as distinct from the initiation of fibrosis which does not produce detectable changes in normal pulmonary function. One important indication of this fact is that the development of clinical

<sup>255</sup> See J.C. McDonald and Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile," p. 8. See also, RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 33-34; and RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, p. 29. See also, Peto, "The Hygiene Standard for Chrysotile Asbestos," at p. 488, where the author has stated that "There are no grounds for assuming linear dose-response for such a generalised progressive disease . . . ."

<sup>256</sup> For example, in his own studies, Dr. J.C. McDonald testified that he finds a linear relationship for both asbestosis and lung cancer and also, although not as good, for gastrointestinal cancer. See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 33-34. Dr. Acheson in his testimony noted that a linear hypothesis fits the data from Rochdale and from New Orleans. See RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, pp. 14-15.

<sup>257</sup> Dr. Finkelstein testified that "... for all practical purposes there is probably an effective threshold below which clinical asbestosis will be unlikely to occur, although there might be the occasional very susceptible individual who could develop problems." See RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 70. Dr. Nicholson testified that "There really is like a threshold for mortality from asbestosis." See RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 66. See also, Peto, "The Hygiene Standard for Chrysotile Asbestos," p. 488; and RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 96.

<sup>258</sup> RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 34.

asbestosis has been almost exclusively limited to occupationally exposed populations where exposures have historically been heavy.<sup>259</sup> There is no evidence that it has ever been caused by asbestos exposure outside the workplace. Even, for example, the extensive study of the household contacts of workers at the Paterson, New Jersey, amosite insulation factory conducted by Dr. Henry A. Anderson et al. revealed minimal parenchymal abnormalities.<sup>260</sup> A total of 679 family members who had lived with workers at the plant during their employment (1941–1954) were all examined 20 or more years after the time of their first exposure.<sup>261</sup> They were essentially healthy when examined; pleural changes predominated and very few had even advanced irregular opacities.<sup>262</sup>

The fact that there have been no cases of asbestosis among those not occupationally exposed to asbestos indicates that the fibrotic process does not progress to the point of clinical manifestation among those with low exposure. We note that the absence of clinical asbestosis among the general public led the U.K. Advisory Committee to suggest “. . . that there may be a threshold level here below which asbestosis is not detectable.”<sup>263</sup>

If those not occupationally exposed to asbestos have no risk of developing clinical asbestosis, what is the risk for those occupationally exposed? Dr. E. Donald Acheson and Dr. Martin J. Gardner, in their report to the U.K. Advisory Committee, “The Ill Effects of Asbestos on Health,” came down in favour of a dose-response relationship without a threshold for chrysotile asbestos within the range of exposures experienced in industry.<sup>264</sup>

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<sup>259</sup>Dr. Finkelstein testified: “I think that asbestosis is not a public health problem . . . I believe that asbestosis is almost exclusively limited to occupationally exposed populations. That’s clinical asbestosis.” See RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 71.

<sup>260</sup>Henry A. Anderson et al., “Asbestosis Among Household Contacts of Asbestos Factory Workers,” *Annals of the New York Academy of Sciences* 330 (14 December 1979): 387–399.

<sup>261</sup>*Ibid.* The study was limited to the 679 household contacts who had lived in a household of an actively employed amosite asbestos factory worker and who themselves had not had an occupational exposure to asbestos or other fibrogenic dust.

<sup>262</sup>See RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, pp. 39–40. Dr. Anderson admitted that while some of these family members are at increased risk, still the likelihood for any one of them to reach the point of disability from asbestos exposure is low.

<sup>263</sup>U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee*, paragraph 103, p. 58.

<sup>264</sup>Acheson and Gardner, “The Ill Effects of Asbestos on Health,” paragraph 191, p. 38. In his evidence before this Commission, Dr. Acheson testified that “One may be less certain that there is no threshold in relation to asbestosis, in view of the fact that although it is known that a very large proportion of the urban population of industrialized Europe and North America have substantial numbers of asbestos fibres in their lungs, they do not at autopsy have evidence of asbestosis. So it may very well be that there is a threshold in respect of asbestosis.” RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 15.

However, at the time their report was written, the control limit for chrysotile asbestos in the United Kingdom was 2 f/cc and the calculation of health risks was predicated on a working life of 50 years, yielding a cumulative dose of 100 f/cc-yrs.<sup>265</sup> What is the likelihood of clinical asbestosis occurring when the cumulative exposure is one-half, one-quarter, or one-fifth of this level?

Dr. Finkelstein has recently examined the cumulative probability of having developed certified asbestosis (that is, being adjudged by the Ontario Workers' Compensation Board to have asbestosis) by the end of the 32nd year from first exposure among 157 long-term employees (15 or more years' employment) of the Scarborough Johns-Manville plant.<sup>266</sup> He found the probability to be 90% among the very heavily exposed, that is, with a cumulative exposure in the range of 200 to 249 f/cc-yrs; falling to 10% among men with a cumulative exposure of less than 50 f/cc-yrs; and to 1% or less around 10 f/cc-yrs. (See Figure 5.1.) These results led Dr. Finkelstein to conclude that "... the major risk at lower exposures will be due to cancer rather than to asbestosis."<sup>267</sup>

Similar findings to those of Dr. Finkelstein were made by Mr. Berry in his examination of 197 workers employed after 1950 and for 10 or more years at Rochdale.<sup>268</sup> Here the average follow-up was shorter, averaging 16 years, but Mr. Berry was looking only at the prevalence of "possible asbestosis," essentially a combined judgement of two physicians as to whether a worker had developed signs attributable to early asbestosis. Average annual exposure for the 197 workers was 5 f/cc and the prevalence of possible asbestosis, 6.6%. This prevalence rate fell to zero or near zero at cumulative exposures of 10 f/cc-yrs.<sup>269</sup>

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<sup>265</sup> British Occupational Hygiene Society, Committee on Hygiene Standards, "Hygiene Standards for Chrysotile Asbestos Dust," pp. 51-52. Using basal rates as the key symptom, the authors of the report concluded that the concentration giving a risk of 1% for an individual working for 50 years in the asbestos industry was estimated to be 2.2 f/cc as measured with the standard membrane filter method.

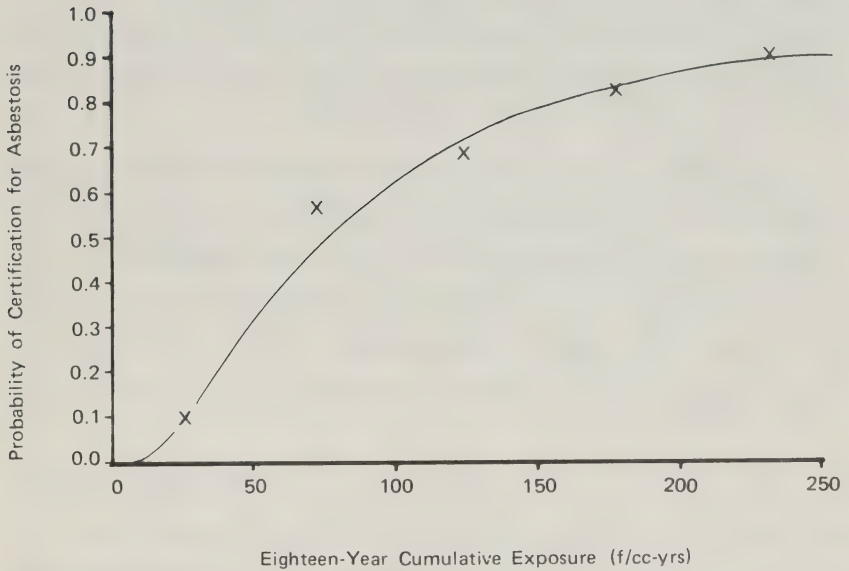
<sup>266</sup> Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," pp. 496-501.

<sup>267</sup> *Ibid.*, p. 501.

<sup>268</sup> Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory."

<sup>269</sup> *Ibid.*, p. 109. The authors noted that the average cumulative exposure of the group first employed after 1950 at Rochdale was 84 f/cc-yrs. In view of their findings, the authors concluded that "... there is no room for complacency about the 2 f/cm<sup>3</sup> standard . . ." The authors also presented data as to the prevalence of certified asbestosis in relation to cumulative exposure. Once again, the average interval from first exposure was 16 years and the maximum was 23 years. For workers in the 50 to 99 f/cc-yrs category, they observed a prevalence rate for certified asbestosis of about 2.5% and for workers in the 100 to 149 f/cc-yrs category, the prevalence rate was 8.5%. By comparison, Dr. Finkelstein's probabilities, evaluated at the end of the 22nd year from first exposure, were 4% and 6% respectively. See Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 501.

**Figure 5.1**  
**The Cumulative Probability of Certification for**  
**Asbestosis (as evaluated at the end of 32 years from**  
**first exposure) Versus the 18-Year Cumulative Exposure\***



**Note:** \* The curve represents the log-normal fit to the data.

**SOURCE:** Murray M. Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," *American Review of Respiratory Disease* 125 (1982): 499 (Figure 3).



An even more recent study of Rochdale has been conducted by the British Occupational Hygiene Society (BOHS). In updating Mr. Berry's work, the BOHS Committee on Asbestos examined 295 men who had commenced employment after 1950 and who had a minimum of 10 years' exposure to asbestos by December 1976.<sup>270</sup> However, the Committee's end point was not "certified asbestosis" or even "possible asbestosis." It was instead whether one of seven defined radiological or clinical adverse effects was present.<sup>271</sup> The Committee found that the probability of any one of these effects occurring at exposures less than 50 f/cc-yrs was 7%, falling to under 2% for cumulative exposures less than 25 f/cc-yrs.<sup>272</sup>

Dr. Weill's earlier findings from his examination of 859 workers employed in two asbestos-cement manufacturing plants in the New Orleans area are also indicative of a cumulative exposure level below which clinical asbestosis would be unlikely to occur, at least in that cohort. Using a conversion ratio of 2 f/cc for 1 mppcf, Dr. Weill suggested that below 100 mppcf-yrs, or 200 f/cc-yrs, diffuse pulmonary fibrosis would not likely be found. Such a level would be equivalent to an exposure of 5 f/cc per year for a working lifetime of 40 years; or an exposure of 8 f/cc per year for a working lifetime of 25 years.<sup>273</sup>

We note also Dr. Enterline's observation that despite the many workers exposed to asbestos in the United States, there have been very few reported deaths from asbestosis since 1950; in fact, between 1950 and 1977, there were 747 reported deaths from asbestosis, with the trend rising until 1972 and then levelling off at an average of approximately 50 per year.<sup>274</sup>

We acknowledge that these data do not establish beyond any doubt the existence of a cumulative exposure level below which clinical asbestosis will not occur. We recognize that among some cohorts studied, even workers in the lower cumulative exposure categories (which correspond more closely

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<sup>270</sup> British Occupational Hygiene Society, "Report from the Committee on Asbestos: A Study of the Health Experience in Two U.K. Asbestos Factories," S.A. Roach, Chairman, *Annals of Occupational Hygiene* 27:1 (1983): 1-55.

<sup>271</sup> Ibid., p. 10, where the seven criteria are set out.

<sup>272</sup> Ibid., p. 1.

<sup>273</sup> See Weill et al., "Lung Function Consequences of Dust Exposure in Asbestos Cement Manufacturing Plants." See also, Hans Weill, "Radiographic and Physiologic Patterns Among Workers Engaged in Manufacture of Asbestos Cement Products: A Preliminary Report," *Journal of Occupational Medicine* 15 (1973): 248-252.

<sup>274</sup> Philip E. Enterline, "Proportion of Cancer Due to Exposure to Asbestos," in *Banbury Report 9: Quantification of Occupational Cancer*, Table 1, p. 21. In his testimony before the Commission, Dr. Enterline observed that at today's much lower exposure levels, clinically disabling asbestosis is much less a matter of concern than it has been historically. See RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 96. Dr. Enterline also observed that in North America there is a long history of reporting of asbestosis cases, so it is unlikely that very many cases have gone unreported. RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 9.

to modern conditions) have died as a result of asbestosis. We recognize too that variations in susceptibility among individuals make it difficult to have any confidence in a no-effect or threshold level. What is a threshold for one person may well not be a threshold for another. Nevertheless, our assessment of the evidence finds us in agreement with Dr. Finkelstein's comment that there is probably an effective threshold below which clinical asbestosis may be unlikely to occur, although there may be occasional cases developing in very susceptible individuals.<sup>275</sup> At low levels of occupational exposure to asbestos the fibrotic process in the lungs, if indeed it can be initiated, will not likely progress to the point of clinical manifestation or even the mildest discomfort. On the basis of the available data, our best judgement as to the lifetime occupational exposure to asbestos at which the fibrotic process cannot advance to the point of clinical manifestation of asbestosis is in the range of 25 f/cc-yrs and below. We are sustained in this view by Dr. Weill's evidence to the effect that as a practising chest physician he now rarely sees asbestosis to a degree which is so functionally severe that it has an important impact on the cardio-respiratory system.<sup>276</sup> In our judgement, asbestosis can be deemed a disease of past high exposure levels and will not occur in workers exposed to the regulated levels of occupational exposure now in force in Ontario.

### **(b) Lung Cancer**

Most epidemiological studies of asbestos workers that have demonstrated an excess lung cancer risk associated with the inhalation of asbestos have produced results consistent not only with a linear relationship between cumulative dose and mortality, but also consistent with the absence of a threshold. We include among these studies that of the Quebec chrysotile miners by Dr. J.C. McDonald et al.; the Charleston, South Carolina, textile workers studied separately by Dr. Dement et al. and Dr. A.D. McDonald et al.; and the Manville factory workers studied by Dr. Enterline.<sup>277</sup> In all of these studies there appears to be a progressive and proportional increase in

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<sup>275</sup>RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 70-71.

<sup>276</sup>RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 43.

<sup>277</sup>See the review of the various studies of asbestos workers in Section B of this chapter. Dr. Acheson testified that from his review of the evidence on lung cancer mortality, the data were consistent with a linear hypothesis and no threshold. See RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, pp. 14-15. Dr. Nicholson testified that none of the studies gives any evidence for a threshold effect of asbestos exposure and all of the studies are best described by the use of a linear dose-response relationship, although in some cases an even more severe relationship, that is, one that would have increased effect at lower exposure, may obtain. See RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 61.

the SMR for lung cancer with increasing dose and no evidence of a threshold level.<sup>278</sup>

This evidence cannot be accepted without some qualification, however, at least in part because of the limitations of the available data base. All of the studies have the intractable difficulty of separating out the effects of cumulative dose from duration of exposure.<sup>279</sup> Mr. Peto has even suggested that the apparently very good correlation between relative risk and cumulative dose found in many published studies actually reflects the correlation between duration of exposure and relative risk, and he points to his own recent study of the Rochdale textile workers as supportive of that proposition.<sup>280</sup> Moreover, there are some studies — for example, the Connecticut friction materials plant only just reported on by Dr. A.D. McDonald and the Scarborough cement workers reported on by Dr. Finkelstein — in which the higher lung cancer mortality rates are found not in the higher but in the lower cumulative exposure categories.<sup>281</sup> Such results are difficult to interpret but may reflect the uncertainty of the available data, including exposure estimates, the possible confounding effects of smoking, and selectivity in employment; or they may simply be attributable to chance.

Another study — Dr. Weill's New Orleans asbestos-cement workers — has produced results that are consistent both with and without the postulation of a threshold.<sup>282</sup> Indeed, in his evidence to this Commission, Dr. Weill suggested that an exposure level that was low enough to prevent

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<sup>278</sup>In "The Hygiene Standard for Chrysotile Asbestos," Mr. Julian Peto observed that a linear relationship for lung cancer is supported by published work, and he referred both to Dr. Enterline's and Dr. J.C. McDonald's data. He noted that, by contrast, "If the duration of brief exposure can be taken as a measure of total dose the results of [the amosite study by Seidman, Lilis, and Selikoff] appear to suggest that for respiratory cancer lower doses may be proportionately more dangerous . . ." (p. 486.) Such apparent non-linearity could be due to selective removal of workers who develop respiratory symptoms, saturation at very high exposures, underestimation of expected numbers, or random error in estimates of dose or duration of exposure. In summing up the various papers on asbestos delivered at the IARC conference in Lyon in 1979, Dr. Weill stated: "In regard to carcinogenic effects, there appeared to be a consensus that the shape of the curve is linear and that no threshold exists." See Hans Weill, "Asbestos — A Summing-Up," in *Biological Effects of Mineral Fibres*, vol. 2, p. 872.

<sup>279</sup>See RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 38–40; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 27.

<sup>280</sup>RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 137. See also, Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," p. 833.

<sup>281</sup>See Sections B.2 and B.4 of this chapter.

<sup>282</sup>Weill, Hughes, and Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing." See also, RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 75–77.



asbestosis would likely be sufficiently low to preclude detectable excess risk for lung cancer.<sup>283</sup> By implication a threshold level for asbestosis might also be a threshold level for lung cancer. Both Mr. Berry and Dr. J.C. McDonald lent support to Dr. Weill's proposition, agreeing that if dust levels were reduced so that asbestosis did not occur then lung cancer would be mainly controlled, although both injected the caveat that it would not be completely controlled and some lung cancer cases would occur.<sup>284</sup>

We do not find it surprising that there is debate about the carcinogenic risk at low exposure levels because lung cancer risks at low doses over a working lifetime have not to date been estimated by observation but rather by extrapolation from observed risks at higher exposure levels.<sup>285</sup> Accordingly, there is no direct evidence of the existence or absence of a threshold for lung cancer. It may arguably be the case that with further inquiry and better information the scientific community will be able to demonstrate that there is a dose level for asbestos for which the body's defence mechanisms are effective or that asbestos acts differently at lower rather than higher doses, thus demonstrating a threshold level for the induction of cancer. At the present time that information does not appear to us to exist. Since a threshold dose level for asbestos-related lung cancer has not been established, we believe it prudent to assume for regulatory purposes that there is none and that any dose may induce lung cancer.<sup>286</sup> This is certainly consistent with the weight of the epidemiological evidence. Several studies, for example, those by Dr. Dement and by Dr. Seidman, clearly show an excess risk of lung cancer mortality at concentrations where asbestosis is not an important cause of mortality.<sup>287</sup> Moreover, our assessment of the epidemiological literature is fortified by two further observations. First, from our understanding of the medical literature, a linear dose-

<sup>283</sup> RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 34-35, 132.

<sup>284</sup> RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p.

41; and RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, pp. 83-84.

<sup>285</sup> Weill, "Asbestos — A Summing-Up," p. 872.

<sup>286</sup> In his article, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part II. Mortality," at pp. 196-197, Mr. Peto suggested that there are three qualitative alternatives: (i) assume that because no excess risk has been demonstrated at levels below, for example, 2 f/cc, one should assume that such levels are virtually safe; (ii) assume that low levels are almost as dangerous as moderate levels until they are proved not to be; or (iii) assume a linear dose-response relationship and legislate accordingly. Mr. Peto rejected the first alternative as too optimistic and the second as too pessimistic and neither supported by biologic knowledge nor epidemiological data. He argued for the third alternative as being the most reasonable and as being a pragmatic proposal, rather than a scientific statement. He suggested that a linear dose-response relationship should be assumed until it is disproved for the purpose of setting a hygiene standard for asbestos rather than to argue that the theories of carcinogenesis that predict non-linearity are necessarily wrong.

<sup>287</sup> See Sections B.3 and B.5 of this chapter. See also, Seidman, Selikoff, and Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," Table 3B, p. 65.



response model without a threshold is biologically plausible and is consistent with current models of carcinogenesis.<sup>288</sup> In this vein, we note Dr. J.C. McDonald's testimony that the dose-response line for lung cancer is one of the most precise estimates of exposure-response in cancer published in any field today.<sup>289</sup> Second, a linear non-threshold model is less likely to underestimate the risks at low doses than any other plausible model.<sup>290</sup> In light of these observations and the strength of the epidemiological evidence, we have no hesitation in proceeding on the basis that there is a dose-response relationship for lung cancer and that it is best described by a linear non-threshold model or, on a graph, by a straight line through the origin. Of course, the slope of that line, or the degree of increase in relative risk for corresponding increases in dose, has varied widely among the different occupational cohorts that have been studied.

### (c) Mesothelioma

The nature of the dose-response relationship for mesothelioma has been less firmly established than that for either asbestosis or lung cancer.

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<sup>288</sup>See Kenny S. Crump et al., "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," *Cancer Research* 36 (September 1976): 2973-2979. In the article, the authors demonstrated that "... under some reasonable assumptions about carcinogenic mechanisms and processes that dose responses will be approximately linear at low doses." (p. 2977.) The authors went on to state that "Virtually all models of carcinogenesis that depict the exposure as affecting an already ongoing process will lead to linearity at low dose . . . . This result then implies that no matter what the biological mechanism we might imagine, if the carcinogen increases some part of the already ongoing process, then we should expect a response to be approximately linear at low dose." (p. 2978.)

<sup>289</sup>RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 14. Dr. J.C. McDonald also testified:

. . . if we imagine that one component of the onset of malignant disease is damage to the nucleus to produce a mutation, each insult to the nucleus of cells, in this case lung cells, will carry, if you like, a finite risk that something will go wrong. So it is reasonable that the dose should be directly related to the probability of this mutation occurring. This will hold up even if there is, if you like, a repair mechanism, because the repair mechanism will still, if you like, have to be dealing with a hazard which is dose-related. (See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 33.)

<sup>290</sup>RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, p. 33; and RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 13. Mr. Berry has noted that there are in addition two main practical objections to basing a hygiene standard on the assumed existence of a threshold: first, the value of this threshold could never be determined from the epidemiological data (Mr. Berry cited by way of example, if at a certain dose there were no cases of disease out of 1,000 individuals at risk, then it could only be stated with reasonable certainty that the risk was less than 3 in 1,000); and second, even if the threshold value were known, it would not be possible to conclude from a limited number of dust measurements that a worker would never be exposed to higher concentrations. See Geoffrey Berry, "Hygiene Standards — Theory and Application," in *Biological Effects of Asbestos*, pp. 145-149. See also, RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 95.

Indeed, it has been suggested that very trivial doses of asbestos (even, some have said, no more than one day's exposure) are capable of inducing the disease and that as a result there is no dose-response relationship for mesothelioma at all.<sup>291</sup> That mesothelioma is associated with low levels of exposure for brief periods of time appears to be based upon isolated anecdotal case reports and upon more systematic case-series reports of mesothelioma arising from non-occupational household or neighbourhood exposures.<sup>292</sup> For example, Dr. Anderson, in his continuing investigation of disease among the household contacts of the amosite asbestos workers at Paterson, New Jersey, has already found 5 cases of pleural mesothelioma, and tracing of these contacts is not yet complete.<sup>293</sup> Dr. Anderson has also indicated that by 1979 there were published reports of over 50 mesothelioma deaths arising from domestic exposure.<sup>294</sup> He has further noted that in those cases arising in non-occupationally exposed individuals, pleural mesotheliomas predominated over peritoneal mesotheliomas.<sup>295</sup>

In the mesothelioma survey conducted by Dr. J.C. McDonald and Dr. A.D. McDonald, embracing 22 countries in the years 1959–1976, the authors identified 230 cases attributable to home or neighbourhood exposure.<sup>296</sup> We are of the opinion that this evidence is not inconsistent with the existence of a dose-response relationship for mesothelioma.

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<sup>291</sup> See RCA Transcript, Submission by Mr. Roy Steinfurth on behalf of the International Association of Heat and Frost Insulators and Asbestos Workers, 8 June 1981, Volume no. 7, p. 44; and Canadian Environmental Law Association, Written submission to the Royal Commission on Asbestos, #45, 17 February 1981, p. 9. In his testimony, Dr. Finkelstein referred to the fact that "One often hears quoted or sees in print the statement that because, you know, someone with one day's exposure has developed mesothelioma, there is no dose-response and this is hazardous at all levels." See RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 91. The witness rejected that view in his testimony. See note 308, *infra*. Mr. Berry testified that "Mesotheliomas are known to occur after incidental exposure, and this has led to several people putting forward the opinion that there was no dose-response relationship." See RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 17. In fact, as Mr. Berry subsequently explained in his testimony, the first data establishing a dose-response relationship for mesothelioma were taken from a report by Newhouse and Berry on the Cape Asbestos East London factory. See Newhouse and Berry, "Patterns of Mortality in Asbestos Factory Workers in London."

<sup>292</sup> See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 92–94. See also, Margaret R. Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," *American Review of Respiratory Disease* 114:1 (July 1976): 211.

<sup>293</sup> Anderson et al., "Asbestosis Among Household Contacts of Asbestos Factory Workers," p. 387.

<sup>294</sup> Henry A. Anderson et al., "Household Exposure to Asbestos and Risk of Subsequent Disease," in *Dusts and Disease*, p. 155.

<sup>295</sup> RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 20.

<sup>296</sup> J. Corbett McDonald and Alison D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," *Preventive Medicine* 6:3 (September 1977): 428–429.

The number of cases occurring among those either occupationally or non-occupationally exposed ought not to be considered without also having regard to the exposure levels involved and the number of persons at risk. For example, the 230 cases identified by the McDonald and McDonald survey occurred among a huge population at risk.<sup>297</sup>

More recent data which have considered these factors strongly support the existence of a dose-response relationship for mesothelioma, a relationship which, like that for lung cancer, is best described by a linear non-threshold model. The first such data came from the Newhouse and Berry study of the Cape Asbestos East London factory where approximately 7% of worker deaths were attributable to mesothelioma.<sup>298</sup> The authors, by dividing their cohort into four rough exposure categories of "severe" or "low to moderate" with more or less than 2 years' exposure, were able to demonstrate a dose-response relationship. (See Table 5.31.) Similarly, the study by Jones et al. of the female gas mask assemblers at Nottingham, England, in World War II (using crocidolite) showed that 2.8% of those workers with 10 to 20 months' exposure contracted mesothelioma, rising to 4.6% for those with 20 to 30 months' exposure, and to 9.8% for those with over 30 months' exposure.<sup>299</sup> In Dr. Finkelstein's mortality study of the Johns-Manville plant, while the numbers are admittedly small, there is a clear trend to an increasing risk of mesothelioma with increasing exposure and the results are compatible with a linear dose-response relationship.<sup>300</sup> We observe also that both animal studies and lung tissue studies evidence that mesothelioma operates in accordance with recognized principles of dose-response.<sup>301</sup> And as both Dr. Paul Kotin and Dr. William J. Nicholson

<sup>297</sup> Ibid. The following countries were included in the study: Australia, Belgium, Canada, Cyprus, Czechoslovakia, Eire, Finland, France, Federal Republic of Germany, German Democratic Republic, Greece, Israel, Italy, Netherlands, South Africa, Spain, Sweden, Switzerland, U.K., U.S.A., U.S.S.R., and West Indies.

<sup>298</sup> Newhouse and Berry, "Patterns of Mortality in Asbestos Factory Workers in London."

<sup>299</sup> See Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," Table 5, p. 646.

<sup>300</sup> See Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," p. 18. See also, Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 142.

<sup>301</sup> Dr. Davis testified that in his animal injection studies he gets very good dose-response relationships for mesothelioma. See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, p. 95. See also, RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, pp. 28-29. See also, Wagner, Berry, and Timbrell, "Mesotheliomata in Rats After Inoculation with Asbestos and Other Materials." The authors, at p. 178, indicated that a varying dose experiment gave results which indicated that the risk of developing mesothelioma at a given time after injection was proportional to the dose. See also, F. Whitwell, J. Scott, and M. Grimshaw, "Relationship Between Occupations and Asbestos-Fibre Content of the Lungs in Patients with Pleural Mesothelioma, Lung Cancer, and Other Diseases," *Thorax* 32 (1977): 383.

Table 5.31  
Mesothelioma Death Rates

Exposure Category and Duration (Years)	Pleura	Peritoneum	Subject-Years	Rate per 100,000 Subject-Years
Males				
Low to moderate				
<2	3	1	12,031	33
>2	3	4	7,500	93
Severe				
<2	6	10	15,428	104
>2	7	12	7,827	243
Laggers				
<2	3	2	7,893	63
>2	1	4	2,690	186
Females				
Low to moderate	1	0	2,066	48
Severe				
<2	8	5	9,538	136
>2	4	3	4,388	360

SOURCE: Muriel L. Newhouse and Geoffrey Berry, "Patterns of Mortality in Asbestos Factory Workers in London," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 57 (Table 5).



told the Commission, there is biologically no reason to doubt that the incidence of mesothelioma is dose-related.<sup>302</sup>

We recognize that the reported deaths from mesothelioma after what appear to have been brief (for gas mask workers) or low (for family contact and neighbourhood cases) exposures have caused considerable public anxiety concerning this disease. We cannot confirm the accuracy or otherwise of the proposition that there are cases of mesothelioma which have occurred from insignificant doses of asbestos. However, we think it unlikely. The doses to which the gas mask workers were exposed appear to have been quite intense, albeit brief, in duration;<sup>303</sup> pictorial evidence and subsequent experiments have indicated that household and neighbourhood exposures where mesothelioma resulted were in fact considerably higher than originally thought and approached, or in many cases were equivalent to, corresponding occupational exposures.<sup>304</sup>

Dr. Davis told the Commission that the notion that mesothelioma occurs at low doses first arose from the documented cases of mesothelioma among those who lived in the vicinity of the Cape crocidolite mines in South Africa and from the cases occurring among the wives of asbestos workers who merely brushed their husbands' overalls when they came home from work at night. Dr. Davis is of the opinion that the wrong conclusion

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<sup>302</sup>See RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 81; and RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 66. Dr. Nicholson indicated that while the data on the dose-response relationship for mesothelioma are very scanty, those data that do exist are completely consistent with a linear dose-response relationship. See also, RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 15 to the same effect. And see Sheers and Coles, "Mesothelioma Risks in a Naval Dockyard," in which mesothelioma incidence rates were correlated both with duration of employment in exposed occupations and the relative severity of exposure of various shipyard jobs. There is also some suggestion of a relationship between the extent of the exposure and the site of a mesothelioma tumour. Dr. J.C. McDonald testified that in a U.K. study of all the cases of mesothelioma known to the Cape Asbestos Company, the heavily exposed individuals had a high proportion of peritoneal mesotheliomas; the victims who were lightly exposed, even to crocidolite, tended to have pleural mesotheliomas. See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 102.

<sup>303</sup>Mr. Peto testified that the mesothelioma cases among the gas mask workers, almost without exception, had very high fibre content in their lungs, so that they were apparently exposed to very high doses over a short period. See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 37.

<sup>304</sup>See RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 16; RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 92-94; RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 106; and RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, pp. 77-81.

has been drawn from this evidence.<sup>305</sup> He has seen pictures which demonstrate that in the past, crocidolite waste was used to surface roads in South Africa and that vehicles driving along these roads produced dust clouds likely as high as in asbestos mines or factories. Further, Dr. Davis is aware of experiments which have shown that exposing overalls to asbestos clouds of the sort that might have occurred years ago and then brushing them produced dust levels in the order of 200 f/cc.<sup>306</sup>

While all of this evidence suggests that the incidence of mesothelioma is dose-related, none of it sustains the proposition that there is a threshold for this disease. We accept this fact and would therefore expect to see a few persons contracting mesothelioma after brief or low exposures. But as Mr. Peto observed in his testimony:

To assume on the basis of anecdotes that people who drilled a hole in an asbestos roof when they were 20, who get mesotheliomas 50 years later due to that exposure, I think it's completely unreasonable. I think it's very unlikely that the effect of brief exposure is more than proportional, and the majority of cases seem to be occurring in people who have had fairly substantial exposure.<sup>307</sup>

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<sup>305</sup> RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 92-94.

<sup>306</sup> Ibid. See also, RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, pp. 91-92. Dr. J.C. McDonald also testified that the concentrations of asbestos fibres in the homes of asbestos workers can be very high because all the soft materials accumulate fibres which may then be shaken out. See also, RCA Exhibit II-57, Tab 8, in RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41: Ross Hunt, "Differences Between the Re-Aerosolability of Settled Airborne Chrysotile and Amphibole Dusts," extracts from reports issued to the Asbestosis Research Council, Cleckheaton, Yorkshire, BBA Group PLC, Industrial Health Unit, 26 May 1982. (Mimeographed.) Mr. Hunt described an experiment wherein membrane samples were fastened to the handles of sweeping brooms, and sweeping operations were carried out using mixtures of crocidolite and chrysotile dust. The dust counts obtained showed that crocidolite became airborne in respirable dimensions very readily, while chrysotile, although airborne, was present mainly in accreted clusters which were not respirable. When cotton overalls were deliberately contaminated with the two fibre types and then subjected to moderately violent patting with the hands, the crocidolite fibres became airborne in respirable fractions as opposed to the chrysotile which flocculated and presented itself as non-respirable fibre aggregates. A room 9 feet square was used to demonstrate the re-aerosolability of crocidolite. Contaminated clothing was shaken in the room and subsequently a broom was used to move the dust from one part to another. Counts of 6 f/cc down to 2 f/cc were obtained over a period of two weeks.

<sup>307</sup> RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 131-132.

And as Dr. Finkelstein commented:

My feeling is that you may certainly develop a mesothelioma with a short exposure, but the risk is substantially less than the risk at higher exposures. . . .<sup>308</sup>

Of course, such cases that do occur tend to stand out, more, for example, than lung cancer, because of the asbestos-specific nature of the disease. On our assessment of the available data, we have no reason to disagree with the conclusion reached in the “Report of the Advisory Committee on Asbestos Cancers to the Director of the International Agency for Research on Cancer” in 1973 that insofar as mesothelioma is concerned, “There is no evidence of a risk to the general public at present.”<sup>309</sup>

## E.4 Age and Time Dependency of the Response

As we have observed earlier in this Report, there is a time interval between the initial exposure to asbestos and the clinical manifestation of the diseases it causes. This time interval is commonly known as latency, and diseases from inhaling asbestos, like diseases from many other agents that cause pneumoconiosis and cancer, are thought to have a very long latency period.<sup>310</sup> These latency periods are difficult to define or measure precisely, but on the basis of observed cases for asbestosis and asbestos-related cancers other than mesothelioma, they are rarely less than 10 years and often more than 20 years. Mesotheliomas appear to have the widest range of latency — again, they rarely occur less than 10 years from the time of first exposure to asbestos, but they can occur as many as 40 years or more from the onset of exposure.<sup>311</sup> In the 21 cases of mesothelioma reported by

<sup>308</sup>RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 91.

<sup>309</sup>“Report of the Advisory Committee on Asbestos Cancers to the Director of the International Agency for Research on Cancer,” in *Biological Effects of Asbestos*, p. 342. See also, RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 74. Dr. J.C. McDonald testified that the contribution of occupational exposure is the main factor affecting the increased rate of mesothelioma in males, and the absence of an increased rate in females implies that non-occupational exposure is probably not having any important effect in North America.

<sup>310</sup>See U.S., Department of Labor, Occupational Safety and Health Administration, “Identification, Classification and Regulation of Potential Occupational Carcinogens,” 29 CFR Part 1990, 45 FR 5002–5296, 22 January 1980 [hereafter “The OSHA Cancer Policy”]. It is stated in “The OSHA Cancer Policy” that “The fourth distinctive characteristic of cancer, the long *latent periods* between exposure and effect, was abundantly documented in the Record. Many witnesses pointed out that there is a long latent period between the initial events in carcinogenesis and the clinical manifestations of the disease. . . .” (p. 5026.)

<sup>311</sup>RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 14–15; Alison D. McDonald, “Mesothelioma Registries in Identifying Asbestos Hazards,” *Annals of the New York Academy of Sciences* 330 (14 December 1979): 441; and Zielhuis, *Public Health Risks of Exposure to Asbestos*, pp. 78–80.



Dr. Finkelstein from Scarborough, the latency period ranged from 14 to 32 years; in all but 3 of these cases death occurred before age 65 and at least 5 deaths occurred while the individuals were in their 40s.<sup>312</sup> By contrast, all 5 cases of pleural mesothelioma among the household members of the Pater-son, New Jersey, amosite asbestos workers occurred 30 or more years from the time of first exposure.<sup>313</sup> And of the 16 cases of mesothelioma among the Quebec chrysotile miners, 4 occurred 30 to 39 years from first exposure; 5 occurred 40 to 49 years from first exposure; and 6 occurred 50 or more years from first exposure.<sup>314</sup>

The obvious effect of latency is that the asbestos-related diseases we observe today are the result of exposures many years ago; correspondingly, whether the exposures of the past few years might lead to asbestos-related disease cannot be known with certainty for years to come.

Recently, a number of experts working in the field of cancer have reconsidered the traditional concept of latency and have viewed the time interval between first exposure and clinical manifestation of the disease from a new perspective. Cancer is a disease that occurs predominantly in the middle and older age groups,<sup>315</sup> and these experts believe that what has been called a latency period is simply a difference in the time between first exposure and the time when the disease most often occurs.<sup>316</sup>

Moreover, in the case of asbestos, the time considerations that apply to lung cancer appear to be quite different from those that apply to meso-thelioma. Recent work, both by Mr. Julian Peto while he was at the University of Oxford and by Dr. William J. Nicholson at the Mount Sinai School of Medicine in New York, suggests that the death rates from mesothelioma appear to rise at an exponential rate from the time since first

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<sup>312</sup>Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," p. 14. This study confirms that 18 of the 21 mesothelioma deaths occurred below the age of 65. An earlier version of the same study presented at the Commission's hearings indicated that of the first 16 deaths from mesothelioma at the Scarborough plant, 5 of the victims were in their 40s at the time of death. See RCA Exhibit II-36, Tab 7, in RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 79; Murray M. Finkelstein, "Mesothelioma Deaths Among Johns-Manville Workers." (Table, Mimeographed.)

<sup>313</sup>RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 32. See also, Anderson et al., "Asbestosis Among Household Contacts of Asbestos Factory Workers."

<sup>314</sup>A.D. McDonald, "Malignant Mesothelioma in Quebec," p. 676.

<sup>315</sup>John Cairns, *Cancer: Science and Society* (San Francisco: W.H. Freeman and Company, 1978), chap. 2, pp. 5-14. See also, Peto, Seidman, and Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," pp. 131-132.

<sup>316</sup>Mr. Peto in fact testified that latency as the term is normally used is not a helpful concept. See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 66-70.



exposure.<sup>317</sup> Mr. Peto's work suggests that mesothelioma death rates rise at a rate between the third and fourth power of time since first exposure; and Dr. Nicholson's work suggests the fifth power of time. What both have demonstrated is that the incidence of mesothelioma rises rapidly the longer the time period since a person is first exposed to asbestos. As a result, the age at which a person is first exposed to asbestos becomes a very significant factor in determining the overall risk of contracting mesothelioma. Utilizing the data from Dr. Selikoff's study of North American insulation workers, Mr. Peto has been able to demonstrate that the risk of contracting mesothelioma is approximately 10 times higher for persons first exposed to asbestos when of school age as compared to persons first exposed when over age 40.<sup>318</sup> This is illustrated in Table 5.32. While the mesothelioma incidence rates appear to be independent of the age at which exposure first took place, the practical result is that the risk of contracting mesothelioma is greater the earlier in life one is first exposed. The magnitude of the risk will still depend on the amount and duration of exposure (and, it may be added, the type of fibre); and where that exposure is minimal, the risk,

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<sup>317</sup> See Julian Peto, Brian E. Henderson, and Malcolm C. Pike, "Trends in Mesothelioma Incidence in the United States and the Forecast Epidemic Due to Asbestos Exposure During World War II," in *Banbury Report 9: Quantification of Occupational Cancer*, pp. 51-69; Peto, Seidman, and Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," pp. 124-135; Julian Peto, "Dose and Time Relationships for Lung Cancer and Mesothelioma in Relation to Smoking and Asbestos Exposure," in *Zur Beurteilung der Krebsgefahren durch Asbest* [Proceedings of the Bundesgesundheitsamt Asbestos Symposium], Berlin: February 1982, bga Schriften MMV Medizin Verlag München, in press, 1983; William J. Nicholson et al., "Cancer from Occupational Asbestos Exposure: Projections 1980-2000," in *Banbury Report 9: Quantification of Occupational Cancer*, pp. 87-108; and RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 36-37, 40-41.

<sup>318</sup> See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 10-12; and Julian Peto, "An Alternative Approach for the Risk Assessment of Asbestos in Schools," report to the U.S. Environmental Protection Agency, 6 April 1981. (Mimeographed.)

Table 5.32

**Predicted Numbers of Mesotheliomas That Would Occur  
Among a Given Number of Men in Each Quinquennium Following  
First Exposure to Asbestos at a Fixed Level, by Age at First Exposure**  
[Incidence Assumed to Increase as (Time Since First Exposure)<sup>3.5</sup>]

Age at Diagnosis	Age First Exposed							
	16-20	21-25	26-30	31-35	36-40	41-45	46-50	51-55
	Median Age First Exposed							
	18½	23½	28½	33½	38½	43½	48½	53½
Predicted Numbers of Mesotheliomas								
25-29	0.04							
30-34	0.17	0.04						
35-39	0.48	0.17	0.04					
40-44	1.07	0.47	0.16	0.04				
45-49	2.02	1.05	0.47	0.16	0.03			
50-54	<i>3.40*</i>	1.97	1.02	0.46	0.16	0.03		
55-59	5.19	3.24	1.87	0.97	0.43	0.15	0.03	
60-64	7.22	4.77	<i>2.98</i>	1.72	0.90	0.40	0.14	0.03
65-69	9.12	6.31	4.17	<i>2.60</i>	1.51	0.79	0.36	0.13
70-74	10.32	7.40	5.12	3.39	<i>2.72</i>	1.23	0.65	0.30
75-79	10.15	7.50	5.39	3.73	2.47	<i>1.55</i>	0.91	0.49
Total below age 80	49.18	32.92	21.22	13.07	7.62	4.15	2.09	0.95

**Note:** \* Figures corresponding to cases occurring in 1974-1978 in men first exposed in 1942 are italicized.

**SOURCE:** Julian Peto, Brian E. Henderson, and Malcolm C. Pike, "Trends in Mesothelioma Incidence in the United States and the Forecast Epidemic Due to Asbestos Exposure During World War II," in *Banbury Report 9: Quantification of Occupational Cancer*, eds. Richard Peto and Marvin Schneiderman ([Cold Spring Harbor, New York]: Cold Spring Harbor Laboratory, 1981), Table 1, p. 53.

albeit greater for exposures earlier rather than later in life, will also be minimal.<sup>319</sup> The time-dependence of mesothelioma appears to provide a reasonable explanation for the observed incidence of the disease in industrial cohorts and also appears to accord with current models of carcinogenesis. Accordingly, we use a model that incorporates the time-dependence (and age-independence) of mesothelioma in our risk assessment found in Chapter 7.

The disease rate of lung cancer among persons exposed to asbestos appears to be quite unlike that of mesothelioma. Rather than being time-dependent, lung cancer rates appear to be age-dependent.<sup>320</sup> The majority of lung cancer deaths, both in smokers and non-smokers, occur after age 50 and over half occur after age 60, irrespective of the time of first exposure.<sup>321</sup> This suggests that the risk of contracting lung cancer is much greater in older groups than in younger groups. Asbestos exposure appears to have the effect of multiplying the risk of lung cancer which exists apart from that exposure; and the risk of lung cancer contributed to by asbestos exposure appears to be virtually independent of the age when that exposure took place and will be simply proportional to cumulative dose.<sup>322</sup> Accordingly, even if a person is first exposed to asbestos quite late in life, the effect of that exposure in terms of the risk of contracting lung cancer may be

<sup>319</sup>In his paper, "An Alternative Approach for the Risk Assessment of Asbestos in Schools," Mr. Peto calculated the mesothelioma risk up to age 80 due to exposure at 1 f/cc for 6 years from age 12, and from exposure to 1 f/cc for 10 years from age 30. On his calculation, the earlier exposure would produce 329.1 total deaths per 100,000 and the later exposure, 107.2 deaths per 100,000. Since asbestos levels in schools are always 1/100 and usually very much less than 1/1,000 of 1 f/cc, the lifelong risk would accordingly be at least 100 and probably more than 1,000 times lower than this calculation and thus is likely to be less than 1 in 100,000, which seems to be negligible. See Table 4, p. 13. In "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," Peto, Seidman, and Selikoff clearly stated that their model does not mean that the risk is unrelated to fibre type and intensity of exposure. Their calculations were based upon the mortality experience of the North American insulation workers exposed to amosite. The authors further indicated that if the contribution to subsequent mortality due to each inhaled asbestos fibre is proportional to the cube of time since inhalation, the death rate following brief exposure would rise as the cube of time since first exposure; the rate due to continuous exposure would rise as the fourth power of time; and the effect of intermediate duration would be well approximated by an exponent of time between 3 and 4.

<sup>320</sup>See Peto, "An Alternative Approach for the Risk Assessment of Asbestos in Schools," p. 1; and RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 16-22.

<sup>321</sup>See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 65.

<sup>322</sup>See RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 27-28. The dose, of course, still remains important with respect to lung cancer as well as mesothelioma. Dose affects the relative risk. The higher the dose, the higher the relative risk. But the dose itself does not affect the latency period. Thus, it is incorrect to suggest that if one reduces exposure, one increases the latency period. A reduction in exposure simply reduces the relative risk of contracting the disease. See *ibid.*, pp. 30-34; and RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 66.

quite substantial because it will have the effect of multiplying the already elevated lung cancer risk that pertains to older age groups. By contrast, the risk of contracting lung cancer at age 20 or age 40 from asbestos exposure is quite negligible because the young have a minimal risk of the disease apart from asbestos exposure.<sup>323</sup> As the model is in accordance with accepted principles of carcinogenesis and is consistent with the epidemiological data, it forms the basis for our risk assessment for lung cancer in Chapter 7.

## F. The Effect of Smoking

It is now a well-accepted fact that cigarette smoking plays a critical role in the incidence of lung cancer.<sup>324</sup> Smoking has been a common habit among working populations including those individual cohorts of asbestos workers that have been subjected to epidemiological study. It therefore becomes important to consider the extent to which the incidence of lung cancer among asbestos workers is attributable to their smoking habits rather than to their asbestos exposure. Beyond that, it is relevant to explore the extent to which smoking affects the risk of contracting the other asbestos-related diseases.

The seminal study on the relationship between smoking and asbestos disease is that of Hammond, Selikoff, and Seidman.<sup>325</sup> These authors obtained complete smoking histories for 8,220 men out of a cohort of 12,051 North American asbestos insulation workers who had 20 or more years of employment. As a control population, the authors used the 73,763 men in the American Cancer Society's prospective cancer prevention

<sup>323</sup>RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 65; and RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 22.

<sup>324</sup>See, for example, Richard Doll and Richard Peto, "Cigarette Smoking and Bronchial Carcinoma: Dose and Time Relationships Among Regular Smokers and Lifelong Non-Smokers," *Journal of Epidemiology and Community Health* 32 (1978): 303-313. See also, Richard Doll and Richard Peto, "The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today," *Journal of the National Cancer Institute* 66:6 (June 1981): 1191-1308. The authors have stated that:

No single measure is known that would have as great an impact on the number of deaths attributable to cancer as a reduction in the use of tobacco or a change to the use of tobacco in a less dangerous way. The principal impact would be on the incidence of cancer of the lung, which by late middle age is more than ten times greater in regular cigarette smokers than in lifelong non-smokers, but a material effect would also be produced on the incidence of cancers of the mouth, pharynx, larynx, esophagus, bladder, probably the pancreas, and perhaps the kidney. (p. 1220.)

<sup>325</sup>E. Cuyler Hammond, Irving J. Selikoff, and Herbert Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 473-490.



study.<sup>326</sup> The authors found that asbestos workers who did not smoke had approximately a 5 times greater risk of dying of lung cancer than the non-smoking control population. Similarly, asbestos workers who did smoke had about a 5 times greater risk of dying of lung cancer than the smoking control population. Smokers were at 11 times greater risk of lung cancer than non-smokers; and the risk for smoking asbestos workers was elevated approximately 55-fold over the non-smoking control population.<sup>327</sup> Table 5.33, which was referred to by several witnesses during our hearings, illustrates these differences.

These rates are applicable to the particular cohort of insulation workers that was studied, and we would not be justified in applying them to other populations with different smoking habits, different levels of asbestos exposure, and different lung cancer risks.<sup>328</sup> Nonetheless, in our judgement, the Hammond study does indicate five relevant findings of more general application concerning asbestos exposure, smoking, and lung cancer. They are as follows:

(i) The relative risk of contracting lung cancer from asbestos is approximately the same for both smokers and for non-smokers. In other words, asbestos multiplies by the same amount the risk of contracting lung cancer for smokers and non-smokers alike.<sup>329</sup> In turn, this evidences the fact that asbestos on its own, in the absence of smoking, is capable of inducing lung cancer, a finding that has been amply confirmed both on the basis of experimental models and other cohort studies.<sup>330</sup>

(ii) While there is a risk of lung cancer for non-smoking asbestos workers, the great preponderance of lung cancers, in absolute terms, occurs in those asbestos workers who have smoked.<sup>331</sup> In the Hammond study, of the 276 asbestos workers who, according to death certificate information, died of lung cancer and whose smoking habits were known, only 4 never smoked regularly and only 4 others smoked only a pipe or cigar. By comparison, there were 152 workers who were current smokers of 20 or more cigarettes a day at the time of their death.<sup>332</sup> Accordingly, while asbestos

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<sup>326</sup>Ibid., pp. 473-475.

<sup>327</sup>Ibid., pp. 486-487.

<sup>328</sup>See RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, p. 21.

<sup>329</sup>See Peto, "Dose and Time Relationships for Lung Cancer and Mesothelioma in Relation to Smoking and Asbestos Exposure," p. 2.

<sup>330</sup>RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), pp. 19-20; and 22 July 1981, Volume no. 21(A), p. 76. See also, J. Corbett McDonald, "Asbestos and Lung Cancer: Has the Case Been Proven?" *Chest* 78:2 (August 1980, Supplement): 374-376; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 64.

<sup>331</sup>RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), pp. 19-20.

<sup>332</sup>Hammond, Selikoff, and Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," Table 7A, p. 486.

**Table 5.33**  
**Age-Standardized Lung Cancer Death Rates\* for**  
**Cigarette Smoking and/or Occupational Exposure**  
**to Asbestos Dust Compared with No Smoking and No**  
**Occupational Exposure to Asbestos Dust**

<b>Group</b>	<b>Exposure to Asbestos?</b>	<b>History Cigarette Smoking?</b>	<b>Death Rate</b>	<b>Mortality Difference</b>	<b>Mortality Ratio</b>
Control	No	No	11.3	0.0	1.00
Asbestos workers	Yes	No	58.4	+ 47.1	5.17
Control	No	Yes	122.6	+ 111.3	10.85
Asbestos workers	Yes	Yes	601.6	+ 590.3	53.24

**Note:** \*Rate per 100,000 man-years standardized for age on the distribution of the man-years of all the asbestos workers. Number of lung cancer deaths based on death certificate information.

**SOURCE:** E. Cuyler Hammond, Irving J. Selikoff, and Herbert Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 487 (Table 8).

exposure does increase the risk of contracting lung cancer in smokers and non-smokers alike, because the absolute risk of lung cancer is so small in non-smokers, the contribution of asbestos in absolute numbers is also small.<sup>333</sup>

(iii) In combination, asbestos exposure and smoking act synergistically in causing lung cancer. In the actual study by Dr. Hammond, the numbers are consistent with a multiplier effect between asbestos and smoking (with death rates of 11.3, 58.4, 122.6, and 601.6 per 100,000 men for non-exposed non-smokers, asbestos-exposed non-smokers, non-exposed smokers, and asbestos-exposed smokers respectively).<sup>334</sup> The significance of the interaction between asbestos and smoking becomes even clearer by expressing the results of the Hammond study in a different fashion. Of the 1,946 observed deaths in the cohort of insulation workers studied, 450 deaths, according to the best available evidence, were caused by lung cancer. Dr. Selikoff, in a separate report, subsequently considered how many lung cancer deaths would have occurred in the cohort absent smoking and/or asbestos.<sup>335</sup> According to his calculations, if there had been asbestos exposure and no cigarette smoking there would have been 44 lung cancer deaths; if there had been no asbestos exposure but cigarette smoking there would have been 94 deaths; and if there had been neither cigarette smoking nor asbestos exposure there would have been only 9 lung cancer deaths.

Mr. Berry's studies in England have similarly indicated that asbestos and smoking combine multiplicatively to produce lung cancer,<sup>336</sup> and the

<sup>333</sup>As Mr. Peto stated in "Dose and Time Relationships for Lung Cancer and Mesothelioma in Relation to Smoking and Asbestos Exposure": "For lung cancer, the relative risk (ratio of incidence to incidence in unexposed individuals of the same age and smoking habits) is more or less independent of age and cigarette smoking, but the absolute risk in the general population is strongly related to both age and cigarette smoking." (p. 2.) See also, RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 73; and J.C. McDonald, "Asbestos-Related Disease: An Epidemiological Review," p. 590.

<sup>334</sup>See Table 5.33 in this chapter.

<sup>335</sup>Irving J. Selikoff, "Two Comments on Smoking and the Workplace," *American Journal of Public Health* 71:1 (January 1981): 92.

<sup>336</sup>See RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, pp. 109-110. See also, Geoffrey Berry, Muriel L. Newhouse, and Mary Turok, "Combined Effect of Asbestos Exposure and Smoking on Mortality from Lung Cancer in Factory Workers," *The Lancet* 2:775 (2 September 1972): 476-479. The authors studied the smoking habits of over 1,300 male and 480 female factory workers and their mortality from lung cancer over a 10-year period. No significant excess was found in workers, whether smokers or non-smokers, with low to moderate asbestos exposure, but among workers who smoked and who were severely exposed the excess was highly significant. The analysis, particularly of the data relating to women where there was a high proportion of non-smokers, supports the multiplicative hypothesis for the action of asbestos and tobacco. See also, Newhouse and Berry, "Patterns of Mortality in Asbestos Factory Workers in London," p. 59.

data from Dr. J.C. McDonald's Quebec chrysotile mining study are compatible with a multiplicative model.<sup>337</sup> Whether an exact multiplier effect will hold for every individual cohort is not overly important. What is important is that there is a very powerful interaction between cigarette smoking and asbestos which produces an effect that is certainly greater than the sum of their individual contributions. This conclusion, drawn from various cohort studies, is also supported by experimental evidence which suggests that asbestos particle clearance in smokers is considerably lower than in non-smokers.<sup>338</sup> This suggests that smoking may impair the clearance of asbestos fibres from the lung. Dr. Kotin testified that chronic smoking over a period of time will initiate a process that results in the death of the ciliated cells, thereby reducing the effectiveness of the important clearance mechanisms in the lung.<sup>339</sup> He also suggested that cigarette smoke would interfere with the other main defence against asbestos fibres, the macrophages. As cigarette smoke is also a material foreign to the body, the same macrophages used to clear asbestos fibres will be needed to clear cigarette smoke.<sup>340</sup> Still, the exact manner in which asbestos and cigarette smoke combine in synergistic fashion to produce lung cancer remains uncertain.<sup>341</sup>

<sup>337</sup>RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 63-64. In fact, Dr. J.C. McDonald suggested that, on balance, his data are more compatible with an additive model than with a multiplicative one. The following table of relative risks illustrates this:

	Asbestos Exposure		
	Little	Moderate	Heavy
Non-smokers	1	2.0	6.9
Moderate smokers	6.3	7.5	12.8
Heavy smokers	11.8	13.3	25.0

SOURCE: J. Corbett McDonald, "Asbestos-Related Disease: An Epidemiological Review," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), p. 590.

In the end, Dr. J.C. McDonald concluded that one can certainly say that there is a very important interaction between smoking and asbestos and it could be anything between additive and multiplicative.

<sup>338</sup>Richard A. Lemen et al., *Workplace Exposure to Asbestos: Review and Recommendations*, prepared by the NIOSH/OSHA Asbestos Work Group, April 1980, DHHS (NIOSH) Publication no. 81-103 (Washington, D.C.: U.S. Department of Health and Human Services and U.S. Department of Labor, November 1980), pp. 27-28. See also, reference made therein to a study by D. Cohen, S.F. Arai, and J.D. Brain, "Smoking Impairs Long-Term Dust Clearance from the Lung," *Science* 204 (4 May 1979): 514-517.

<sup>339</sup>See RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 62-68. Thus, the cigarette smoker would have a higher retention of asbestos fibres than would the non-smoker. If an individual is a cigarette smoker, his ability to clear asbestos fibres from his lungs will be impaired and he will accordingly have a greater effective dose of asbestos in his lungs.

<sup>340</sup>*Ibid.*, p. 73.

<sup>341</sup>*Ibid.*, p. 75. See also, John E. Craighead and Brooke T. Mossman, "The Pathogenesis of Asbestos-Associated Diseases," *The New England Journal of Medicine* 306:24 (17 June 1982): 1451.



(iv) It is evident that of the two variables, cigarette smoking and asbestos exposure, the former appears to have the more powerful effect on the incidence of lung cancer.<sup>342</sup> For example, in the Hammond study, smoking increased the incidence of lung cancer by a factor of 11; asbestos, by a factor of 5.<sup>343</sup> While we have no reason to doubt this observation at the present time, a word of caution is in order. Dr. Enterline testified that for a long period of time Dr. Selikoff did not observe any deaths among his non-smoking insulation workers, although he now clearly does. Dr. Enterline compared this to a similar pattern he has observed among uranium miners where there is also a synergistic effect between radiation and smoking. According to Dr. Enterline, it now appears that non-smoking uranium miners are beginning to experience a very high incidence of lung cancer, and he speculates that what we may be observing in uranium miners is the disease incidence further downstream. He has suggested that we may also witness a downstream incidence of lung cancer among non-smoking asbestos workers.<sup>344</sup> At the present time that suggestion remains in the realm of speculation.

(v) To the extent that asbestos-exposed persons stop smoking, their risk of death from lung cancer, like that of non-asbestos exposed persons who stop smoking, decreases quite considerably.<sup>345</sup> Again, utilizing the North American insulation workers data, the risk of lung cancer mortality for those workers who had previously smoked but stopped in 1967 was approximately one-third that of their workmates who continued to smoke over the next 10-year period.<sup>346</sup>

Smoking appears to have a different relationship to asbestosis than it does to lung cancer. Smoking can clearly initiate lung cancer; it does not initiate the fibrotic process that leads to asbestosis. Further, there are some studies such as that of Dr. Weill which evidence no relationship at all

<sup>342</sup>RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 84; and RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, pp. 53-55.

<sup>343</sup>That smoking is a more powerful variable than asbestos exposure is also illustrated in the study by Dr. J.C. McDonald et al. of the Quebec chrysotile miners. See note 337, *supra*.

<sup>344</sup>RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 88-90.

<sup>345</sup>Dr. Nicholson testified that if one looks at the mortality rate for lung cancer in individuals who stop smoking cigarettes, one finds that it can fall dramatically over a 10-year period to perhaps a quarter or even less than that which it would have been had one continued to smoke, whatever one's cigarette habit had previously been. After 5 years of cessation of cigarette smoking, the risk of death from lung cancer decreases to perhaps one-half or one-third what it would have been had one continued to smoke cigarettes. RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 55-56.

<sup>346</sup>Irving J. Selikoff, "Asbestos-Associated Disease," in *Public Health and Preventive Medicine*, 11th ed., ed. John M. Last (New York: Appleton-Century-Crofts, 1980), chap. 13, p. 579.

between smoking and the progression of the fibrotic process.<sup>347</sup> However, the weight of the available data indicates that cigarette smoking may affect the progression of the fibrotic process, both in terms of whether clinical asbestosis becomes manifest and of its subsequent rate of progression. The Hammond study found that asbestosis death rates were nearly 3 times higher for heavy smokers than for non-smokers, prompting the authors of the study to conclude that cigarette smoking greatly increases the risk of an asbestos worker dying from asbestosis or from fibrosis in combination with emphysema resulting from smoking.<sup>348</sup> Mr. Berry considered the effect of smoking on the incidence of clinical asbestosis at Rochdale. For men first employed after 1950 with at least 10 years' employment by 1966, very little evidence of asbestosis was found other than in smokers.<sup>349</sup> (See Table 5.34.) On the basis of the data from Rochdale and similar data from the Royal Navy dockyard workers studied by Rossiter, Berry concluded that smokers had a higher risk of developing clinical asbestosis during their lifetime than non-smokers, and his results indicated that for a non-smoker with less than 100 f/cc-yrs exposure, the risk of developing the clinical manifestation of asbestosis was minimal.<sup>350</sup>

The data pertaining to the possible association between asbestos and smoking in relation to laryngeal cancer and cancer of the gastrointestinal tract are scanty. It does, however, appear that most of those in whom carcinoma of the larynx is associated with asbestos exposure have been smokers.<sup>351</sup> Dr. Selikoff's work suggests that the increase in cancer of the esophagus among asbestos workers occurs only in smokers. No such association was demonstrated, however, for cancer of the stomach, colon, or rectum.<sup>352</sup>

In striking contrast to lung cancer, there is no evidence to demonstrate any relationship whatsoever between cigarette smoking and mesothelioma.

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<sup>347</sup>RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 25. See also, Weill et al., "Lung Function Consequences of Dust Exposure in Asbestos Cement Manufacturing Plants," p. 96.

<sup>348</sup>Hammond, Selikoff, and Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," pp. 484-485.

<sup>349</sup>RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, pp. 23-26; and Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," p. 104.

<sup>350</sup>Charles E. Rossiter and Geoffrey Berry, "The Interaction of Asbestos Exposure and Smoking on Respiratory Health," *Bull. europ. Physiopath. resp.* 14 (1978): 202; and Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory."

<sup>351</sup>RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, pp. 20-21.

<sup>352</sup>*Ibid.* See also, Selikoff, "Asbestos-Associated Disease," p. 577.

Table 5.34  
The Association Between Smoking, Age,  
and Signs of Asbestosis

Employment Group and Smoking Habit	Age Group	Numbers of Men				Small Radiological Opacities*
		Total	Crepitations	Possible Asbestosis	Certified Asbestosis	
First employed before 1951	{ -54 55 +	11	0	0	1	0
		18	7	6	2	6
	{ -54 55 +	37	7	5	2	6
		115	42	34	19	47
First employed after 1950	{ -44 45-54	26	0	0	0	1
		17	0	0	0	0
	{ 55 + -44	12	0	0	0	1
		32	0	0	0	1
Other smokers and ex-smokers	{ 45-54 55 +	51	8	5	4	10
		57	18	8	6	15

Note: \* Profusion 1/10 or more.

SOURCE: Geoffrey Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," *British Journal of Industrial Medicine* 36 (May 1979): 104 (Table 7).

Indeed, all available data and all expert opinions support the conclusion that the incidence of mesothelioma is entirely independent of smoking.<sup>353</sup>

## G. What are the Health Effects of Peak Exposures?

High exposures of brief or intermittent duration can and often do occur in some parts of the asbestos industry, particularly in construction, maintenance, demolition, and repair work.<sup>354</sup> This has led us to inquire whether or not these more intense exposures of asbestos — or short, sharp bursts as we referred to them during our hearings — may carry with them a disproportionate health risk. Is a construction worker who, for example, has a total asbestos exposure of 100 f/cc-yrs but who accumulated this exposure in short yet high doses at greater risk than a factory worker who accumulated the same total exposure in continuous and lower doses (and generally over a longer period of time)?

Unfortunately, the epidemiological studies of asbestos workers are of qualified assistance in answering this question. Even for those cohorts where exposure measurements have been available, they have not differentiated between short, intense exposures and more continuous but lower exposures. Consequently, the epidemiologist has been left with only a single average or cumulative figure. Despite the lack of direct data, we nonetheless have a concern on the basis of scattered evidence and on the basis of what is known about the clearance mechanisms in the lung that peak exposures may well produce a disproportionate amount of disease. That concern was raised by a number of the witnesses who appeared before us. For example, Drs. Becklake, Enterline, Kotin, and Weill all suggested that excessive amounts of asbestos dust inhaled in any one period of time may overload the lung's clearance mechanisms, thereby increasing the amount of asbestos

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<sup>353</sup>RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, p. 45; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 70. See also, Peto, "Dose and Time Relationships for Lung Cancer and Mesothelioma in Relation to Smoking and Asbestos Exposure," p. 2; and Hammond, Selikoff, and Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," p. 487.

<sup>354</sup>See, for example, RCA Transcript, Evidence of Dr. William J. Nicholson, 30 June 1981, Volume no. 15, p. 90.



fibres that are retained within the body compared with the retention that takes place at continuous but lower exposures.<sup>355</sup>

Moreover, there does appear to be some epidemiological evidence, indirect in nature, to support the notion that short, sharp bursts may carry a disproportionate health risk. In Dr. Enterline's earlier study of the Manville factory retirees, excess mortality for all causes of death among maintenance-service workers was 24.3%, whereas for production workers it was only 10.7%; for lung cancer, excess mortality for maintenance-service workers was 328.6%, while for production workers it was 73.7%.<sup>356</sup> Dr. Enterline suggested that the intermittency and possible severity of maintenance workers' exposure may well account for this difference.<sup>357</sup> This observation must, as Dr. Enterline pointed out, be treated with caution because of the possible confounding effect of fibre type — only 26% of the production workers in Dr. Enterline's cohort were exposed to amosite and/or crocidolite, compared to 71% of maintenance workers.<sup>358</sup> Support for Dr. Enterline's suggestion may be found in the results of Dr. Weill's study of the asbestos-cement pipe factory at New Orleans where the

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<sup>355</sup>RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 46–47; RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 41–42; RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 36; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 62–63. Dr. Davis testified that he attempted to examine the effects of intermittent, high asbestos exposures (peak dose levels) in his animal experiments. The findings of his study appeared to refute the idea that short periods at very high exposure levels to asbestos result in a much higher lung burden of retained dust than might be expected from the greater density of the "peak dust clouds." However, in the experimental situation, it was impossible to examine the effects of the realistic differential between ordinary and peak exposures that one sees in the human situation because it is necessary that the ordinary exposure of animals be relatively elevated. In his experiments, Dr. Davis had only a fivefold difference between the peak and the ordinary exposure of his animals, whereas in factories the overall dust levels are sufficiently low that a peak dose caused by, for example, a temporary machine defect could easily be 100 times higher than the ordinary level. See John M.G. Davis et al., "The Effects of Intermittent High Asbestos Exposure (Peak Dose Levels) on the Lungs of Rats," *British Journal of Experimental Pathology* 61 (1980): 272–280; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 103–104.

<sup>356</sup>Enterline, DeCoufle, and Henderson, "Mortality in Relation to Occupational Exposure in the Asbestos Industry," pp. 901–902.

<sup>357</sup>*Ibid.*, p. 900. See also, RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 41–42.

<sup>358</sup>Enterline, DeCoufle, and Henderson, "Mortality in Relation to Occupational Exposure in the Asbestos Industry," p. 900.

maintenance workers exposed intermittently to crocidolite had a substantially higher risk of lung cancer than those in the plant steadily exposed to crocidolite.<sup>359</sup>

A further piece of evidence that also lends support to the observation that peak exposures have particularly adverse health effects is the experience of the North American insulators which has been studied by Dr. Selikoff and his colleagues since the early 1960s. As we have already stated, these insulators were, and are, members of the International Association of Heat and Frost Insulators and Asbestos Workers, primarily employed in the building trades. Here too, the extensive use of amosite may well be a contributing factor to the disease incidence of workers. Moreover, as there is an absence of actual exposure information, there are no data to assess the effects of different exposures.<sup>360</sup> Yet given the nature of the trades in which these workers were engaged, it is not unreasonable to expect, and indeed Dr. Selikoff indicated, that they often experienced short, intense exposures.<sup>361</sup> We cite in Chapter 9 of this Report evidence as to how high these exposures can be — for example, 20 to 100 f/cc in spraying insulation on buildings.<sup>362</sup> The health effect of such peak exposures does offer one possible explanation of the very high cancer incidence among the insulators.

We recognize that this limited evidence by no means firmly establishes the adverse health effects of short, sharp bursts. Indeed, it may be argued that the excess disease incidence observed in maintenance, repair, and insulation workers in some cohorts is due not to intermittent, high exposures but to the fact that these jobs are by their nature dustier and the cumulative

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<sup>359</sup>Weill, Hughes, and Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," pp. 352-353. The SMR for lung cancer among those workers with intermittent exposure to crocidolite in the pipe plant was 304, and the SMR for lung cancer among those workers with steady employment in the pipe plant with crocidolite exposure was only 155. RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 62-63.

<sup>360</sup>On the basis of direct measurements during recent years and data from other studies using similar processes, historical levels to which the insulators were exposed have been estimated at between 10 and 15 f/cc for commercial and industrial construction and between 15 and 20 f/cc for marine construction. See Nicholson, "Criteria Document for Swedish Occupational Standards: Asbestos and Inorganic Fibers," pp. 39-41. In their most recent published article, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," Selikoff, Hammond, and Seidman have stated that "... analysis of available data, including reconstruction of work situations and extrapolation to the past of observations made more recently, suggest that insulation workers would have been exposed to dust levels of 4-12 [f/cc]. . . ." (p. 92.)

<sup>361</sup>Selikoff, Hammond, and Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," p. 93; and Nicholson, "Criteria Document for Swedish Occupational Standards: Asbestos and Inorganic Fibers," p. 39. Dr. Nicholson suggested that the exposures could be extremely high and could range, for example, for 2- to 5-minute concentrations of asbestos, from 50 to 100 f/cc.

<sup>362</sup>See Chapter 9, Section B.1 of this Report.

exposure of workers in these jobs is simply greater; or it may be due, as we have indicated, to their exposure to crocidolite and amosite. We have no way of knowing what the true situation is. Nor does the available evidence tell us anything about how high one might expect these peak exposures should be in order for them to have a differential impact on health. With the limitations of the evidence in mind, we nonetheless conclude that short, sharp bursts of exposure do present a special health risk to workers, one that our regulatory system ought to have regard to, in the control of asbestos exposures both in fixed and non-fixed workplaces.

## **H. Is There a Special Health Risk to Children from Asbestos Exposure?**

One of the concerns that prompted the appointment of this Commission was the possible health hazard to children from exposure to asbestos in Ontario schools. Whether such a hazard in truth exists will of necessity very much depend upon the level and extent of exposures in the school system. This subject we pursue in detail in Chapters 9 and 10. Suffice it to say here that, in general, asbestos exposure levels in the Ontario school system are very low.

In this chapter we pursue a different question and that is whether children are more susceptible than adults to the health effects of asbestos exposure. If such is the case, then whatever are the measured levels of exposure in schools and whatever is the relevant dose-response relationship, we would, out of prudence, have to take particular account of this susceptibility in formulating our recommendations.

From our review of the medical evidence and from our questioning of the various witnesses who appeared before us, we could find no substantive support for the proposition that the lungs of young children are in and of themselves more susceptible than those of adults to asbestos-related diseases.<sup>363</sup> Indeed, there is evidence from animal experiments that might well suggest a contrary conclusion.<sup>364</sup> Despite this lack of evidence, we cannot exclude the biological possibility that children have less immunity to asbestos-related diseases than adults. This possibility arises because the cellular turnover rates in children are generally higher than those in adults. In a growing organ this may well mean much higher rates of mutational changes

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<sup>363</sup>RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 111; RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 122; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 64.

<sup>364</sup>RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 66–67.



of the type thought to initiate the development of malignant tumours.<sup>365</sup> We note that Dr. Acheson, in his oral testimony, suggested we would do well to take into account the possibility that children might be more susceptible than adults in an immunological sense. Indeed, he suggested that there may be a general susceptibility of both young and aged to carcinogenic substances.<sup>366</sup>

Indirect support for this hypothesis may perhaps be found in the work of Wasserman et al. who examined some 30 cases of mesotheliomas reported in the literature in which the tumour developed in infancy or childhood.<sup>367</sup> While such cases are admittedly rare and their association with asbestos-exposed parents by no means certain, Wasserman found two conspicuous characteristics of these mesotheliomas: there was a very high degree of malignancy, the majority dying less than a year from the onset of symptoms, and the latency period from first possible exposure was very short, the maximum being 14 years.<sup>368</sup> The rapidity of the process at least suggests that when children do contract mesothelioma, their immunity to the disease is somewhat less than that observed in adults.

Quite apart from these rare cases of childhood mesothelioma, there are a small number of clearly documented cases of mesothelioma occurring in adults which have been attributed to exposure to asbestos in childhood (invariably the exposure has been traced either to the home or to the neighbourhood of asbestos factories or to waste disposal sites). For example, Dr. J.C. McDonald testified that as a result of his own inquiries into the incidence of mesothelioma in North America based on cases ascertained through pathologists, he found 5 mesothelioma deaths in persons whose fathers were asbestos workers. At least 3 of the workers were employed in the Quebec mines. Death in these 5 persons occurred at ages 31, 32, 41, 42, and 51, thus tending to confirm that exposure to asbestos took place in childhood.<sup>369</sup>

These cases of mesothelioma attributable to childhood exposure do not of themselves indicate any greater susceptibility to asbestos on the part

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<sup>365</sup>RCA Transcript, Evidence of Mr. Julian Peto, 30 July 1981, Volume no. 25(B), p. 125; and RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, pp. 89-91.

<sup>366</sup>RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 26. In its written submission to this Commission, the American Industrial Hygiene Association, Ontario Local Section, suggested that there is some evidence that a child's lungs may preferentially retain long, thin fibres. See American Industrial Hygiene Association, Ontario Local Section, Written submission to the Royal Commission on Asbestos, #11, 12 January 1981, p. 7.

<sup>367</sup>M. Wasserman et al., "Mesothelioma in Children," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 253-257.

<sup>368</sup>*Ibid.*, p. 254.

<sup>369</sup>RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 109-110.



of children, but they do serve to emphasize the importance of the age of first exposure to asbestos. As we have discussed earlier in this chapter, the incidence of mesothelioma appears to rise at a rate between the third and fourth power of time since first exposure. And while this incidence rate is of itself independent of the age at which exposure first took place and in that sense is inconsistent with the notion that there is an age-related susceptibility to mesothelial tumours, the result is that exposures earlier in life carry with them a greater risk than exposures later in life.<sup>370</sup> This is attributable not to any greater biological susceptibility of the young but to the time-dependence of mesothelioma. Even if we set aside this notion, the relatively long latency period (often in excess of 30 years) that seems characteristic of the mesotheliomas occurring among those exposed to asbestos outside the workplace leads to the common sense conclusion that the earlier in life the exposure, the more likely that mesothelioma will occur during a normal lifespan.

There is a further and perhaps a related consideration that deserves mention. There is the possibility that the residence time of asbestos fibres in the lung is an important determinant of the adverse health effects of asbestos. Several witnesses who appeared before us suggested that earlier exposures may be more important in causing disease than later exposures because the longer the fibre might remain in the body, the greater the opportunity to induce adverse effects.<sup>371</sup> Mr. Berry has gone so far as to construct a number of sophisticated models of dose-response that take this factor into account.<sup>372</sup> The potential importance of the residence time of fibres in the lung seems to us plausible, although admittedly at the present time its actual significance is still largely a matter of hypothesis and speculation.

Taken together these various considerations might suggest a slightly higher risk of mesothelioma for children than for adults. But, at the same time, we emphasize that there is no evidence to suggest that the exposure levels to which children have been subjected in asbestos-containing schools or elsewhere, any more than the exposure levels to which others in the general public have been subjected, have posed a health risk. Children have

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<sup>370</sup>RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 122; and RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 57.

<sup>371</sup>See RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, pp. 25–27; Berry et al., “Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory,” pp. 105–109; and Berry and Lewinsohn, “Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part I. Morbidity,” pp. 187–191. See also, RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 66; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 59–60.

<sup>372</sup>Berry and Lewinsohn, “Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part I. Morbidity”; and Berry et al., “Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory.”

been exposed to asbestos in schools in North America for at least 30 years. If the hypothesis that mesothelioma is time-dependent is correct, and if school asbestos exposure were a health risk, we would have expected a number of non-occupational cases of mesothelioma to develop (apart from cases arising from known domestic or neighbourhood exposures). The absence of such cases is strongly indicative of the fact that school exposures are so low that they are not a health problem. Indeed, based on the incidence of mesothelioma among the North American insulators, Mr. Julian Peto has calculated that the cumulative risk of mesothelioma up to age 80 for children exposed to asbestos in schools for 6 years from age 12 onwards is likely to be less than one in 100,000; in other words, negligible.<sup>373</sup>

## I. What is the Health Risk from the Ingestion of Asbestos Fibres?

The principal method by which asbestos fibres enter the body is through inhalation. It is the inhalation of asbestos fibres which has been responsible for the disease incidence found in asbestos workers. But asbestos fibres may also enter the body through ingestion, either in food, beverages, drinking water, or drugs. Anyone in the general population from time to time may well ingest asbestos fibres from one of these sources. For that matter, asbestos workers may be prone to ingest asbestos fibres where food is eaten on the job and has been contaminated by the workplace environment.

It is therefore critical to consider whether there is a health risk from the ingestion of asbestos fibres. Of course, part of the answer to this question may turn on the sizes of asbestos fibres in food and drink and the amount of asbestos actually ingested. This we discuss in Chapter 11 where we provide detailed information on the sizes of fibres in food and drink. Here our inquiry is limited to considering whether there is any evidence that the ingestion of asbestos fibres is capable of producing a human health risk. If there is such a risk, it is likely to be related to malignancies of the gastrointestinal tract. Several cohorts of asbestos workers have indeed demonstrated an excess risk of gastrointestinal cancer from asbestos exposure. And while that excess risk has not been consistent from cohort to cohort — the Rochdale and Charleston textile workers, the New Orleans

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<sup>373</sup>See Peto, "An Alternative Approach for the Risk Assessment of Asbestos in Schools"; and see note 319, *supra*. Dr. Acheson testified that there is no evidence in the United Kingdom of mesothelioma having occurred as a result of exposure to asbestos in schools. See RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, pp. 28-29. Dr. Anderson testified that he was not aware of any mesotheliomas attributable to school asbestos exposure. See RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 60. See also, RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 117.

asbestos-cement workers, and the Nottingham gas mask workers being among the working populations where there was no excess gastrointestinal cancer — there is still a considerable body of evidence which points to an association between asbestos and cancer of the esophagus, stomach, colon, and rectum, albeit a much weaker association than, for example, between asbestos and lung cancer.<sup>374</sup>

Equally significant, that association arises from asbestos exposure through inhalation. There is, to our knowledge, no evidence that the direct oral ingestion of asbestos by itself is capable of producing an excess risk of gastrointestinal cancer. Admittedly, epidemiological methods are not overly sensitive to this issue — first, because it is difficult to find an appropriate control group for comparison purposes and second, because gastrointestinal tumours are much less specifically related to asbestos exposure than, for example, mesothelioma.<sup>375</sup> Yet the fact remains that at the present time the suggestion that eating or drinking asbestos fibres poses a health risk is not supported by any substantial evidence. Indeed, there are three pieces of evidence, apart from actual ingestion levels, that suggest a contrary conclusion. One is that, as Dr. Eric J. Chatfield has indicated, the sizes of fibres actually ingested in food or beverages or drinking water appear to be very small, with a mean length of 2 microns or less.<sup>376</sup> By comparison, as we have seen, the weight of the evidence indicates that it is the longer fibres, 5 or perhaps 8 or more microns in length, which are most hazardous from a health point of view.

A second piece of evidence is the general conclusion that emerges from the various studies that have considered asbestos concentrations in municipal water systems. While recognizing the limitations of epidemiology in addressing this issue, the fact remains that generally there do not appear to be any adverse health effects even where asbestos levels in water systems are thought to be elevated. A recent study by Toft et al. investigated mortality rates and asbestos levels in the drinking water systems of 71 Canadian municipalities and concluded there was no significant relationship between these levels and gastrointestinal cancer.<sup>377</sup> A similar conclusion was reached by Dr. Chatfield after analyzing asbestos levels in the drinking water supply systems of 18 municipalities in Ontario.<sup>378</sup> More recently, Dr. Jack Siemiatycki assessed the mortality experience of the general population in

<sup>374</sup> See Chapter 2, Section D.4 and Sections B and C.5 of this chapter.

<sup>375</sup> RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 95.

<sup>376</sup> RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 97.

<sup>377</sup> Peter Toft et al., "Asbestos and Drinking Water in Canada," *The Science of the Total Environment* 18 (1981): 71–89.

<sup>378</sup> See RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 89–92; and Eric J. Chatfield and M. Jane Dillon, *A National Survey for Asbestos Fibres in Canadian Drinking Water Supplies*, 79-EHD-34 (Ottawa: Health and Welfare Canada, Environmental Health Directorate, 1979), pp. 43–44.



the mining area of Quebec.<sup>379</sup> He noted that Thetford Mines has one of the highest levels of asbestos fibre concentrations among North American drinking water systems and that levels at Asbestos, which has had a filtration system since the 1950s, are also relatively high and would have been higher prior to the 1950s. Table 5.35 shows mortality from selected causes for female residents of Asbestos and Thetford Mines for the period 1966–1977. Despite the fact that the drinking water in Asbestos and Thetford Mines may be as much as 100 times more polluted with chrysotile asbestos than the drinking water in other North American and European cities, the author could find no statistically significant excess mortality from this environmental exposure.<sup>380</sup> Indeed, of the several studies of the health risks from drinking water, only the report by Conforti et al. of the water supply system in the San Francisco Bay area even suggests a relationship between ingested asbestos and gastrointestinal cancer and the relationship is weak.<sup>381</sup> Only a fraction of the many analyses performed by Conforti et al. suggested a correlation of asbestos to cancer, and the authors noted that confounding factors such as smoking, occupation, and alcohol consumption may be important but were not allowed for in the study.

A final piece of evidence is the animal data, the bulk of which tends to demonstrate not merely the absence of malignant tumours, but an absence of asbestos fibres altogether in the gastrointestinal tract of rats following substantial feeding of asbestos.<sup>382</sup> For example, Dr. Davis has carried out a number of animal ingestion experiments on rats. He has found no evidence of asbestos retention within the gut, no sign of cell penetration or damage to the intestinal mucosa, and no evidence at all of gastrointestinal tumours. By contrast, Dr. Davis has pointed out that there is plenty of evidence of asbestos fibres in the gastrointestinal tract of rats following inhalation experiments.<sup>383</sup>

This animal evidence must be viewed with some caution. Although the weight of the animal data appears to be consistent with the findings made by Dr. Davis, a few experiments have indicated that small amounts of

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<sup>379</sup> Jack Siemiatycki, "Health Effects on the General Population (Mortality in the General Population in Asbestos Mining Areas)," in *Proceedings of the World Symposium on Asbestos*, Montreal, Quebec: 25–27 May 1982 (Montreal, P.Q.: Canadian Asbestos Information Centre [1983]), pp. 337–348.

<sup>380</sup> *Ibid.*, p. 342.

<sup>381</sup> Paul M. Conforti et al., "Asbestos in Drinking Water and Cancer in the San Francisco Bay Area: 1969–1974 Incidence," *Journal of Chronic Diseases* 34 (1981): 211–224.

<sup>382</sup> RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 115.

<sup>383</sup> RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 97–100; R.E. Bolton and J.M.G. Davis, "The Short-Term Effects of Chronic Asbestos Ingestion in Rats," *Annals of Occupational Hygiene* 19:2 (November 1976): 121–128; and J.M.G. Davis, R.E. Bolton, and J. Garrett, "Penetration of Cells by Asbestos Fibers," *Environmental Health Perspectives* 9 (1974): 255–260.



**Table 5.35**  
**Mortality, from Selected Causes, in Asbestos**  
**and Thetford Mines Compared to Quebec Province,**  
**Females, 1966-1977**

<b>Cause</b>	<b>O</b>	<b>E</b>	<b>O-E</b>	<b>L.C.L.*</b>	<b>O/E</b>	<b>U.C.L.*</b>
All causes	1,130	1,274.6	-144.6	0.84	0.89	0.94
All cancers	289	318.1	-29.1	0.81	0.91	1.02
Digestive cancer	117	110.7	6.3	0.88	1.06	1.28
Respiratory cancer	23	21.5	1.5	0.68	1.07	1.61
Other respiratory disease	30	51.8	-21.8	0.39	0.58	0.83

**Note:** \*95% confidence limits. ["L.C.L." means lower confidence limit; and "U.C.L." means upper confidence limit.]

**SOURCE:** Jack Siemiatycki, "Health Effects on the General Population (Mortality in the General Population in Asbestos Mining Areas)," in *Proceedings of the World Symposium on Asbestos*, Montreal, Quebec: 25-27 May 1982 (Montreal, P.Q.: Canadian Asbestos Information Centre [1983]), Table 6, p. 348.

asbestos following feeding may be found in intestinal tissues and have suggested an association between ingestion and gastrointestinal tumours.<sup>384</sup> Further, at the present it is not entirely clear as to whether the animal data are even applicable to humans for we are not certain as to how fibres reach the gastrointestinal tract or as to the mechanism by which asbestos apparently induces alimentary tract tumours in humans. We conclude on the basis of the evidence that the inhalation rather than the ingestion of fibres is responsible. One possibility is that asbestos fibres may reach the gastrointestinal tract by being coughed up and swallowed following inhalation.<sup>385</sup> Penetration of the gastrointestinal tract after swallowing would be far less likely since the fibres would tend to be "flushed" through the system. Another possibility suggested by Dr. Davis and more consistent with the animal data is that asbestos, after inhalation (but not ingestion), has the ability to move around the body by the lymphatic channels to various sites, including that of the alimentary tract.<sup>386</sup> However, Dr. Alexander C. Ritchie, Professor of Pathology at the University of Toronto, expressed serious reservations about this possibility in his testimony before the Commission. Dr. Ritchie had no doubt that asbestos fibres can find their way to the gastrointestinal tract following inhalation (and, if anything, he thought the bloodstream a more likely route), but he candidly observed that we really do not know how they get there.<sup>387</sup>

Accepting the uncertain state of medical knowledge on this matter, we cannot exclude completely the possibility of adverse health effects following the direct ingestion of asbestos. However, we deem it most unlikely. First, we believe it unlikely that asbestos fibres penetrate the gastrointestinal tract following ingestion. Second, even if they do, the fibres one ingests from non-occupational sources are very small, much shorter than the length we consider to be hazardous. Accordingly, it is reasonable to conclude that at present the evidence fails to indicate any increased risk of alimentary tract tumours following the direct ingestion of asbestos fibres.

<sup>384</sup>A number of the animal studies have been reviewed in World Health Organization, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, pp. 58–60. See also, Davis, "The Use of Animal Inhalation Experiments in the Study of Asbestos Bioeffects," pp. 569–570.

<sup>385</sup>Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," p. 138.

<sup>386</sup>RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 100–101.

<sup>387</sup>RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, pp. 14–19, 31–33. Dr. Ritchie testified that if one considers the hypothesis that fibres can travel to various sites of the body by the lymphatic channels, there are a number of problems. First, the lymph flow from the stomach to the lung is from the stomach upwards, so that one would have to imagine these inert asbestos fibres somehow swimming against the flow. Even if one allows that the fibres could proceed against the normal direction of the flow, if one takes the ordinary view that the most hazardous fibres are the long, thin ones, then these fibres would have to travel in vessels that are not much bigger than the fibres themselves, and certainly much narrower than 10 microns in length.











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